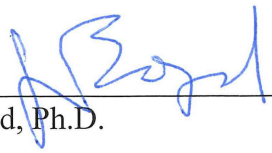
EXHIBIT C22

IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF NEW JERSEY

IN RE: JOHNSON & JOHNSON TALCUM POWDER PRODUCTS MARKETING, SALES PRACTICES AND PRODUCTS LIABILITY LITIGATION ***THIS DOCUMENT RELATES TO ALL CASES***	**MDL NO. 16-2738 (FLW) (LHG)**

EXPERT REPORT OF JEFF BOYD, PHD FOR GENERAL CAUSATION *DAUBERT* HEARING

Date: February 25, 2019



Jeff Boyd, Ph.D.

I. BACKGROUND AND QUALIFICATIONS

I am professor (with tenure) and chair of the Department of Human and Molecular Genetics and professor of Obstetrics and Gynecology, as well as associate dean for Basic Research and Graduate Programs at the Herbert Wertheim College of Medicine at Florida International University. I also serve as associate deputy director, Translational Research and Genomic Medicine, at the Miami Cancer Institute of Baptist Health South Florida. I am founding director of the Center for Genomic Medicine at the Miami Cancer Institute.

I received my bachelor's degree at Duke University and my master's and Ph.D. degrees in toxicology and biochemistry at North Carolina State University, and completed my postdoctoral training in environmental pathology at the Lineberger Comprehensive Cancer Center of the University of North Carolina at Chapel Hill. Following that, I served on the faculty (as a section head of Gynecologic Pathobiology) of the National Institute of Environmental Health Sciences, National Institutes of Health. I then joined the University of Pennsylvania as an associate professor, Division of Gynecologic Oncology, within the Department of Obstetrics and Gynecology, with a joint appointment in the Department of Genetics. From 1997-2006, I worked at Memorial Sloan-Kettering Cancer Center in New York City, where I was director of the Gynecology and Breast Research Laboratory in the Department of Surgery, and director of the Diagnostic Molecular Genetics Laboratory in the Department of Medicine. While there, I was promoted to full member (professor) with tenure-of-title. I left Sloan-Kettering to become vice president of Oncology and Research and director of the Anderson Cancer Institute at the Memorial University Medical Center in Savannah, GA. I also held appointments as professor in the Departments of Obstetrics and Gynecology, Surgery, Medicine, and Division of Basic Medical Sciences, as well as assistant dean for Research at the Mercer University School of Medicine - Savannah. From 2008-2015, immediately prior to taking my positions in Miami, I was a tenured professor and held the Robert C. Young, MD, Chair in Cancer Research at Fox Chase Cancer Center in Philadelphia, where I also served as Senior Vice President, Chief Scientific Officer, and Chief of the Division of Molecular Pathology. In addition, I was founding director of the Cancer Genome Institute.

My research focuses on the genetics and molecular genetics of gynecologic and breast cancers. I have been supported by more than $25 million in grants from the National Institutes of Health or peer-reviewed NIH-equivalent grants, and have served as principal investigator for a National Cancer Institute Specialized Program of Research Excellence grant in ovarian cancer. Additional awards include Distinguished Cancer Scholar from the Georgia Cancer Coalition (2006) and the Rosalind Franklin Award for Excellence in Ovarian Cancer Research from the Ovarian Cancer National Alliance (2015). I have authored or co-authored more than 200 articles, reviews, book chapters and editorials on the molecular and genetic bases of gynecologic or breast cancers, and been invited to present more than 150 lectures on these topics throughout the world. I have served as a peer reviewer in many capacities, including as a standing member of scientific review groups of the National Institutes of Health, the Department of Defense cancer research program, and the American Cancer Society, and as an editorial board member for seven scientific and clinical journals. I have also served as an ad hoc peer reviewer for approximately 45 scientific and clinical journals. Among my many committee and board

memberships, I served as chair of the Scientific Advisory Committee for the Ovarian Cancer Research Fund (Alliance) for nine years, and am currently a member of the Board of Directors for the Society of Gynecologic Oncology. My current research interests include the histogenesis (cell of origin) of ovarian carcinoma, the comprehensive genomic characterization of ovarian cancer stem cells, and the genomic basis of diethylstilbestrol (DES)-induced carcinogenesis of the cervix and vagina of women exposed to DES in utero.

II. SCOPE OF REPORT

I was asked to opine on Dr. Ghassan Saed's expert report based on my experience as a molecular biologist and cancer researcher, and in particular, whether this research supports the biological plausibility of plaintiffs' theory that perineal talc use causes ovarian cancer. All of the opinions in this report are stated to a reasonable degree of scientific certainty. I am being compensated at the rate of $600 per hour for my work on this matter and $1200 per hour for deposition and other testimony.

III. BACKGROUND ON OVARIAN CANCER

Ovarian cancer is a term that embraces several closely-related malignancies. Of most relevance here is epithelial ovarian carcinoma (EOC), which comprises several histological subtypes that together account for approximately 90% of all cases of "ovarian cancer." These subtypes include serous, endometrioid, clear cell and mucinous EOCs. Although the histogenesis (cell of origin) of these cancers remains relatively poorly understood, it has been established that the pathogenesis of the distinct subtypes is not entirely overlapping. For example, a proportion of serous EOCs are now believed to arise in the fallopian tube, while some proportion of clear cell and endometrioid EOCs are believed to arise from implants of endometriosis on the ovary. It should also be noted that from a clinical perspective, carcinomas of the ovary, fallopian tube and primary peritoneal lining are generally treated identically (when matched for stage), in both surgical and medical contexts, and demonstrate a very similar clinical course. Hereafter in this report, the term "ovarian cancer" will be used as defined above.

Among the few accepted significant risk factors for ovarian cancer are rare inherited genetic mutations that affect certain genes, including *BRCA1* and *BRCA2*, which are estimated to substantially increase the lifetime risk of developing ovarian cancer to as high as 40% or 20%, respectively.[1] Additionally, through genome-wide associational studies (GWAS), certain other common genetic variants have been correlated with an increased risk of ovarian cancer, although these variants are associated with a substantially smaller lifetime relative risk of ovarian cancer.[2] Overall, genetic predisposition is currently believed to be associated with approximately 20% of

[1] Kuchenbaecker KB et al., *Risks of Breast, Ovarian, and Contralateral Breast Cancer for BRCA1 and BRCA2 Mutation Carriers.* JAMA (2017) 317(23):2402-16.

[2] Pharoah PD et al., *GWAS meta-analysis and replication identifies three new susceptibility loci for ovarian cancer*. Nat Genet. (2013) 45(4):362-70.

all ovarian cancers.[3] It is very important to recognize that ovarian cancers associated with genetic predisposition as well as those (approximately 80%) that occur "sporadically" are all associated with the acquisition and accumulation of mutations affecting multiple cancer-related genes. So-called "hereditary cancers" differ only in the sense that the first rate-limiting genetic mutation is inherited, rather than acquired. In this sense, all ovarian cancers (and indeed all cancers generally) represent a genetic disease. Multiple mutations affecting multiple genes are required for a normal cell to progress to a malignant tumor cell, regardless of the tissue of origin. The causes of these "somatic" genetic mutations acquired in the organ in which a cancer ultimately develops remain largely unknown for ovarian cancer and most other cancers. Exceptions include a strong association between chronic inhalation of tobacco smoke and lung cancer, and prolonged exposure to ultraviolet-irradiation (sunlight) and skin cancer. Even for these examples, however, it is important to note that never-smokers develop lung cancer and that individuals with very low lifetime exposures to sunlight develop melanoma. Possible mutagenic mechanisms in ovarian and other cancer types include unknown environmental exposures and pure chance. Indeed, one prominent cancer molecular geneticist recently posited that most cancer cases may simply be attributable to bad luck – genetic mutations resulting from chance errors in the ordinary replication of the cellular genome (3.3 billion base pairs per cell) whenever one cell divides into two.[4] If such mutations occur in certain critical genes that affect elements of the cancer cell phenotype, then tumorigenesis may ensue.

The limitations on our understanding of the causes and prevention of ovarian cancer persist notwithstanding decades of intense research efforts in this field. Underscoring these difficulties, a randomized controlled clinical trial involving more than 200,000 apparently well women attempted to assess the viability of ovarian cancer screening over the course of more than a decade. The trial was recently concluded, but shed little light on potential paths forward in identifying ovarian cancer in its earliest and potentially curable stages. As the authors summarized in the published results of this clinical trial, "[f]indings from this trial suggest that for 641 women screened annually using the multimodal strategy for 14 years, one ovarian cancer death is prevented."[5] This disappointing result characterizes the challenges that remain in the area of ovarian cancer research, especially in the areas of etiology and prevention.

IV. PLAINTIFFS' EXPERTS HAVE NOT SHOWN THAT THEIR PROPOSED MECHANISMS FOR OVARIAN CARCINOGENESIS ARE PLAUSIBLE

Plaintiffs' experts propose that talc causes inflammation, which leads to cancer, or that inflammation causes oxidative stress, which damages DNA, which results in cancer. These

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[3] Walsh T et al., *Mutations in 12 genes for inherited ovarian, fallopian tube, and peritoneal carcinoma identified by massively parallel sequencing*. Proc Natl Acad Sci USA (2011) 108(44):18032-7; Norquist BM et al., *Inherited mutations in women with ovarian carcinoma*. JAMA Oncol. (2016) 2(4):482-90.

[4] Tomasetti C & Vogelstein B, *Variation in cancer risk among tissues can be explained by the number of stem cell divisions*. Science (2015) 347:78-81.

[5] Jacobs I et al., *Ovarian cancer screening and mortality in the UK Collaborative Trial of Ovarian Cancer Screening (UKCTOCS): a randomized controlled trial*. Lancet (2016) 387:945-56.

explanations are simplistic, speculative and lack sufficient scientific support to be deemed plausible. All suffer from the same flaw to various degrees: they depend on large leaps of faith connecting one process to another. My focus, however, is on Dr. Saed's report and the underlying study he conducted, which purportedly found that talc causes an oxidative stress response that is associated with an increased ovarian cancer risk.

As set forth below, Dr. Saed's report layers speculation upon speculation. The gap between his research (which is itself filled with many methodological flaws, described below) and elucidating the origins of ovarian cancer is very large. At most, if his research had been conducted in a reliable manner, it would show that placing relatively large amounts of talc on cell lines *in vitro* can alter the expression of certain genes, change the rates of cell proliferation and apoptosis, and increase the secretion of CA-125. But these observations have no bearing on whether ordinary use of talc in a woman's underwear (or perineal area) can cause ovarian cancer, which remains a speculative theory for which plaintiffs have offered no rational scientific support.

A. Study Design Issues

Use of DMSO as Solvent: Dr. Saed determined that he needed to apply talc through a liquid medium to the cells he wished to treat. But talc is poorly soluble in water, so he apparently chose DMSO (dimethyl sulfoxide), a "universal" solvent, in which to dissolve the talc. Dr. Saed apparently believed that he was controlling for the effects of DMSO by treating a control group of cells with the same solvent (but without talc dissolved in it).[6] But he apparently paid no heed to recent research that has called into question whether the use of DMSO as a solvent can alter the effect of the treatment and skew the results.[7] In other words, while a DMSO-only control can theoretically control for the effects of DMSO by itself, it cannot control for the possibility of an interaction between DMSO and talc or DMSO and the cells that could, in and of itself, alter the effect that talc would otherwise have on the cells (if any). Dr. Saed's failure to evaluate this possibility renders most of his results (those involving exposure of cells to talc) unreliable.

Determination of Talc Dosage: Dr. Saed used a very highly concentrated talc solution – 500 mg of talc per 10 ml of DMSO.[8] He then applied relatively enormous doses of talc – from 5 to 100 µg/ml – directly to the treated cells.[9] This represents a far greater talc exposure than human ovarian cells would ever be subjected to under normal physiologic conditions – including as a result of regular perineal use of talcum powder. Indeed, the evidence that ***any*** talc can reach the ovaries from external perineal use is weak.[10] Dr. Saed never estimated the amount of talc he

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6 Saed Dep. Vol. I 117:4-119:10.

7 *See* Hall MD et al., *Say no to DMSO: Dimethyl sulfoxide inactivates cisplatin, carboplatin and other platinum complexes*. Cancer Res. (2014) 74(14):3913-22.

8 Saed Rep. at 14.

9 *Id.*

10 International Agency for Research on Cancer, *Monographs on the Evaluation of Carcinogenic Risks to Humans* Vol. 93: Carbon Black, Titanium Dioxide, and Talc 411 (2010) ("[T]he evidence for retrograde transportation of talc to the ovaries of normal women is weak" and animal studies "showed no evidence of retrograde transport *(cont'd)*

believes would reach the ovary or the fallopian tubes as a result of perineal dusting, despite being directly asked,[11] and other aspects of his deposition testimony support the conclusion that such an anatomical journey would prove improbable for talc particles. In attempting to explain why talc would not produce inflammation and cancer in the intervening areas of the female reproductive anatomy, for example, Dr. Saed repeatedly referred to the "wash" of bodily fluids that would expel particulate matter.[12] Dr. Saed contrasted this protective mechanism to that of the ovaries, which he claims have no mechanism for removing foreign particles.[13] But the logical conclusion of this argument would be that the same mechanisms of expulsion of talc from areas of the female reproductive tract distal to the ovaries (vagina, cervix, uterus, fallopian tubes) should also prevent talc from otherwise migrating – like a salmon upstream – through this wash of bodily fluids, eventually reaching the ovaries.

Even accepting that talc could reach the ovaries to some extent, however, I am aware of no research suggesting that an amount approaching the quantities involved in Dr. Saed's study would ever reach the fallopian tubes or ovaries, and Dr. Saed appears to admit as much.[14] As such, Dr. Saed failed to show that the dose range he used in his studies is applicable to human exposure levels and any subsequent physiological sequela.

Moreover, Dr. Saed's report does not articulate any reason for selecting such high doses, much less any reason why he believes a study using these mega-doses is likely to produce data relevant to carcinogenesis in humans. At his deposition, Dr. Saed suggested that he initially treated cells with an even larger dose of 1000 µg/ml, but found that this dose simply killed the cells, precluding the ability to measure any biological response, and that he, therefore, selected the lower, but still very high, doses reported in his report and manuscript.[15] This is an inappropriate methodology for selecting an appropriate dose range for experiments designed to test the effect of a xenobiotic (foreign chemical or substance, naturally-occurring or otherwise) on cultured human cells *in vitro*, especially when the goal is to provide evidence that such an exposure is directly linked to carcinogenesis in humans.

A fundamental tenet of toxicology is that any chemical or substance, including those generally considered completely safe or inert (for example, food or beverage ingredients, or substances that humans consume or otherwise contact routinely), will almost certainly elicit a measurable biological or physiological response from cells or organisms that are exposed *in vitro* or *in vivo*,

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of talc to the ovaries"). *See* Henderson WJ et al., *Talc and carcinoma of the ovary and cervix*. J Obstet Gynaecol Br Commonw. (1971) 78(3):266-72 (finding no relationship between perineal talc use and ovarian talc burden); Heller DS et al., *The relationship between perineal cosmetic talc usage and ovarian talc particle burden*. Am J Obstet Gynecol. (1996) 174(5):1507-10 (same).

[11] Saed Dep. Vol. I 233:8-234:5.

[12] *Id.* 166:1-2.

[13] *Id.* 165:11-166:2.

[14] *See id.* 233:11-234:1.

[15] *Id.* 55:3-12.

respectively, to any such xenobiotic when administered at an extremely high, i.e., non-physiologic, dose. That said, such biologic responses, e.g., changes in gene expression or cell proliferation, may not necessarily be associated with a "toxic" outcome, e.g., cell death or neoplastic transformation. If one is testing the hypothesis that exposure to a specific xenobiotic is plausibly linked to carcinogenesis in humans, especially if the model system is human cells cultured *in vitro*, it is only logical that the appropriate experimental design would employ a dose range compatible with an equivalent physiologic exposure *in vivo*, if the intent is to argue that the biological responses seen *in vitro* are somehow related to the carcinogenic process *in vivo*. Since it is impossible to know what level of talc, if any, may actually reach the fallopian tubes and ovaries of a woman exposed to hygienic doses of talc applied in the perineal region, the only recourse an experimentalist has in the design of such a study is to employ as large a dose range as necessary in order to elicit measurable biological perturbations. This describes, in essence, an experimental approach of convenience.

It should now be self-evident that this entire experimental design is fundamentally flawed in several respects, in terms of linking the results of these experiments to talc-induced human ovarian carcinogenesis. First and foremost, lower doses more compatible with a physiologic exposure to talc in the human female reproductive tract were not used in these experiments, even if it were possible to determine what significantly lower dose range that may be. Second, the biological perturbations observed in cultured cells exposed to high doses of talc cannot be reliably extrapolated to such biological responses *in vivo*, which is why animals (typically mice or rats) are used in studies designed to predict the human carcinogenic potential of one or another xenobiotic. Finally, absent the malignant transformation of human cells cultured *in vitro* (utilizing several assays traditionally employed to approximate malignant transformation in this context) following exposure to high doses of talc, the rather non-specific biological responses observed in Dr. Saed's experiments cannot be interpreted to conclude that talc exposure causes ovarian cancer *in vivo*. At most, the only conclusion that may be reasonably made from these experiments is that exposure to extremely high doses of talc results in the biological perturbation of human cells cultured *in vitro*,[16] a result that is entirely expected based on well-established principles of toxicology. Several of the problematic experimental issues discussed above will be expanded upon below.

Inadequate Control Experiments: Dr. Saed's studies do not adequately address his hypothesis that there is a biological mechanism linking exposure to talc (a hydrated magnesium silicate compound consisting of magnesium, silicon and oxygen – all of which are found at one or another concentration in the human body, and are in fact considered "essential elements") to ovarian carcinogenesis because Dr. Saed failed to perform additional control experiments designed to test whether other particulate compounds, such as, for example, cornstarch (a powdered carbohydrate derived from the endosperm of corn kernels) or a particulate compound more chemically similar to talc, such as finely ground beach sand (silicon dioxide) produced the same results. Such experiments testing the potential biological effects of other particulate compounds like talc could have been used to determine whether his findings were driven by

[16] Saed Rep. at 14.

some quality that is unique to talc, or rather its particulate form generally, the characteristics of which are shared by many other compounds.

Specifically, in his investigation, talc was dissolved in DMSO and added to cultured cells as an experimental condition.[17] Changes in the levels of RNA and protein expression in these cells were then measured by qPCR (quantitative polymerase chain reaction) and ELISA (enzyme-linked immunosorbent assay) techniques and compared with levels found in cells treated with DMSO only.[18] Dr. Saed concluded that differences in RNA and protein expression between the talc-treated and DMSO-only-treated samples were evidence of an "oxidative stress" response induced by talc exposure.[19] Overlooked, however, was the possibility that these differences were the result of high-dose particulate exposure generally, and not to talc exposure specifically.

A properly designed experiment would have included a condition(s) where cultured cells were treated with at least one, and preferably several, additional non-talc compounds suspended in DMSO. Such control experiments would help an investigator discern the baseline RNA and protein expression level changes that occur in response to addition of particulate matter generally to cultured cells. Dr. Saed testified that the inclusion of such a condition would have been feasible.[20] He admitted that he did not know whether the addition of an inert substance, such as corn starch, to the cell cultures would have yielded the same RNA and protein expression changes that he observed in talc-treated cell cultures.[21] When confronted with the issue of exclusion of such control experiments, Dr. Saed erroneously concluded that inert substances could not cause a similar oxidative stress response profile because the "untreated" cells exposed to DMSO only "didn't show that."[22] The manner in which cultured cells respond to the addition of DMSO alone has no bearing on how they may respond to the addition of DMSO containing a suspended inert particulate substance other than talc.

The failure to include such control experiments to measure potential "oxidative stress responses" to inert particulate substances is a fatal flaw with respect to the veracity of the investigative power of the aforementioned studies to establish a cause and effect relationship between talc exposure and a cellular oxidative stress response. Dr. Saed's only defense to this fundamentally flawed experimental design was that he "tested several fold."[23] However, repeating the same flawed experiment several times cannot overcome this underlying methodological flaw.

Dr. Saed's experiments neither contradict nor support his hypothesis that there is a biological mechanism(s) through which talc may induce an oxidative stress response in cultured human

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[17] Saed Dep. Vol. I 273:10-14.

[18] Saed Rep. at 14-15.

[19] *Id.* at 14-18.

[20] Saed Dep. Vol. I 274:5-9.

[21] *Id.* 273:16-25.

[22] *Id.* 272:20-273:2.

[23] *Id.* 272:14-19.

cells. He merely showed that there are changes in the expression levels of specific RNA and protein molecules that differ between cells treated with DMSO and cells treated with DMSO containing talc. As such, Dr. Saed's studies offer no support for his opinion regarding the biological mechanism by which talc allegedly causes an oxidative stress response in cultured cells *in vitro*, and much further, ovarian carcinogenesis *in vivo*.

Cell lines: There are serious methodological concerns with respect to the types of human cells that were used in Dr. Saed's experiments. Four distinct categories of primary cells or established cell lines were used: 1) The EL1 cell line, derived from human spleen and classified as a monocyte/macrophage cell type; 2) "Normal ovarian epithelial" cells – it may be inferred from Dr. Saed's laboratory notebook and the commercial source of these cells (Cell Biologics) that they are "human primary ovarian epithelial cells derived from normal human ovary tissue"; 3) The FT33 cell line, described by the commercial source as "immortalized human fallopian tube epithelial cells"; and 4) Three human ovarian carcinoma cell lines, SK-OV-3, A2780, and TOV-112D, which are, by definition, derived from human ovarian carcinomas.[24] All three of the ovarian carcinoma cell lines are originally from the American Type Culture Collection; the latter two are described as having been derived from endometrioid ovarian adenocarcinomas, and the SK-OV-3 cell line was derived from ovarian carcinoma ascites (histologic subtype unknown).[25]

It is not at all clear why one would conduct experiments related to xenobiotic-induced ovarian carcinogenesis using a cell line (EL1) derived from the monocyte/macrophage lineage, a white blood cell type involved in the adaptive immunity process. It is similarly unclear why one would conduct such experiments using human ovarian carcinoma cell lines (SK-OV-3, A2780, and TOV-112D); if an experimentalist is testing the hypothesis that exposure of human ovarian cells to a potential carcinogen leads to biological effects related to the tumorigenic process, why would cell lines that are derived from ovarian carcinomas represent an appropriate model? These cells, *ipso facto*, represent the ultimate culmination of the tumorigenic process, and would be expected to possess myriad biological and somatic genetic differences compared to "normal" ovarian epithelial cells. Stated simply, the approach of testing a hypothesis as to how cancer may be experimentally induced, *using cancer cells*, is seriously unsound.

B. Misinterpretation of Results

CA-125 Findings: Dr. Saed reports an increase in cellular release of the CA-125 protein following talc treatment and claims that this "highlight[s] the implications of the pro-oxidant states caused by talc. . . ."[26] This is a confusing assertion because Dr. Saed does not identify the "implications" that increased CA-125 expression purportedly "highlight[s]." If he intends to suggest that increased CA-125 secretion is suggestive of ovarian carcinogenesis, however, then he misunderstands the clinical use of serum CA-125 protein measurements.[27] The FDA-

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[24] Saed Dep. Vol. I, Ex. 1 at SAED000001 (Expert Report Notebook Files).

[25] *Id.*

[26] Saed Rep. at 18.

[27] Notably, in his deposition, Dr. Saed admitted that that he does not know the clinical significance of CA-125. Saed Dep. Vol. I 248:25-250:2.

approved use of measuring serum CA-125 levels is in the context of a "biomarker" to monitor response to ovarian cancer treatment.[28] Although such measurements have also been tested experimentally for decades in an effort to detect ovarian cancer at an early stage, the specificity and sensitivity of serum CA-125 levels in this context are unacceptably low, and the assay is neither useful nor approved for this purpose.[29] Increased serum CA-125 levels have been reported in "benign conditions such as endometriosis, pregnancy, ovulatory cycles, liver diseases and congestive heart failure, as well as in infectious disease such as tuberculosis."[30] Serum levels of CA-125 are also elevated in non-ovarian cancers, such as "breast cancer, mesothelioma, non-Hodgkin lymphoma, gastric cancer, and leiomyoma and leiomyosarcoma of gastrointestinal origin."[31] Therefore, any increase in CA-125 levels observed by Dr. Saed is not necessarily indicative of malignant conditions, much less malignant risk. Because increased CA-125 expression can reflect any number of causes, physiologic states, or conditions other than ovarian cancer, its use as a detection tool is highly disfavored and is considered ineffective from a clinical perspective. Nor does it play any role in ovarian cancer causation. Therefore, any effect that exposure to talc may have on cellular release of CA-125 is irrelevant to the question whether it plays any role in causing ovarian cancer.

Some of the utility of CA-125 as a biomarker does stem from the fact that CA-125 secretion can increase with the onset of ovarian cancer. As discussed, however, CA-125 secretion is highly non-specific and increases are more frequently unrelated to ovarian cancer. Furthermore, clinical use of CA-125 as an early detection marker for ovarian cancer is typically accompanied by a transvaginal sonography.[32] Even then, "reports suggest that sensitivity of early stage disease is limited."[33] If CA-125 is not even a reliable biomarker for the *onset* of ovarian cancer *in vivo*, it is doubtful that CA-125 can be a reliable biomarker for the *increased risk* of onset of ovarian cancer *in vitro*. To the extent that an increase in CA-125 secretion is sometimes associated with ovarian cancer, Dr. Saed still has not shown that CA-125 is a cancer precursor, rather than an effect of such cancer.

These opinions are generally shared by Reviewer #1, who provided a critique of Dr. Saed's manuscript following submission to *Gynecologic Oncology*. The Reviewer writes that, "The significance of this study would be greatly enhanced if a mouse model corroborated the cell line findings. In this reviewer's opinion, the cell line studies alone and the increase in CA-125 while intriguing are not sufficiently convincing."[34]

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[28] Saed Rep. at 18 (citing Jelovac D & Armstrong DK, *Recent progress in the diagnosis and treatment of ovarian cancer*. CA Cancer J Clin. (2011) 61(3):183-203).

[29] *See* above reference to UKCTOCS clinical trial.

[30] Scholler N & Urban N, *CA125 in Ovarian Cancer*. Biomark Med. (2007) 1(4): 513-523 (internal refs. omitted).

[31] *Id.* at 517 (internal refs. omitted).

[32] *Id.*

[33] *Id.*

[34] Saed Dep. Vol. II, Ex. 35 at 2, Gynecologic Oncology Email dated Sept. 19, 2018 re: GYN-18-1020: Final Decision ("Gynecologic Oncology Decision").

Finally, the conclusion stated in the Abstract and elsewhere in the manuscript by Fletcher *et al*. (rejected by *Gynecology Oncology* and under review or perhaps in press at *Reproductive Sciences*), stating that, "Talc exposure also resulted in a significant increase in inflammation as determined by increased tumor marker CA-125," is incorrect and misleading.[35] There was no direct measurement of inflammation in the cultured cells, and a correlation of increased CA-125 secretion with inflammation is speculative at best.

Cell Proliferation and Apoptosis Findings: Dr. Saed claims that he has "shown conclusively that talcum powder . . . enhance[s] cell proliferation, and inhibit[s] apoptosis in EOC cells," as well as in "normal cells, including surface ovarian epithelium, fallopian tube, and macrophages."[36] At his deposition, he took this claim further, asserting that cell proliferation "is an indirect measure of the beginning of [neoplastic] transformation."[37] None of this is correct, and Dr. Saed's attempt to equate cell proliferation with cancer development is profoundly unscientific. As noted above, the lack of appropriate control experiments undermines the specificity of his findings to talc powder, making it impossible to issue such a "conclusive[]" claim. In fact, cell proliferation is a natural response to stress, meaning that this result would be expected to follow many cell treatments *in vitro* and would not remotely be unique to exposure to large doses of talc suspended in DMSO.

In addition, it is unclear why these findings are significant since Dr. Saed testified that there are no studies showing that increased cell proliferation and decreased apoptosis are associated with ovarian cancer risk.[38] The findings also seem irrelevant because Dr. Saed was not aware of any studies showing that these cellular responses are present in any tissue in women who use talc.[39] Nor am I. Regardless, Dr. Saed's broad characterization of these properties as an "oncogenic phenotype"[40] is not consistent with scientific knowledge.

First, cell proliferation is a regular process in tissue homeostasis, and does not indicate that a normal cell has transformed into a cancer cell. Dr. Saed acknowledged this when he explained that "temporary or initial induction of proliferation [] is a normal response of all normal cells to agents."[41] Dr. Saed does not explain in his report why his findings are not simply a typical cellular response to the introduction of a foreign agent, such as talc, in cell culture. Furthermore, according to his lab notebooks, the furthest data collection time point in Dr. Saed's investigation was 72 hours after treatment with talc. At best, Dr. Saed's study provides a snapshot of the

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[35] Saed Dep. Vol. I, Ex. 7 & 8 at 2 (Fletcher NM, Harper AK, Memaj I, Fan R, Morris RT, Saed GM, *Molecular basis supporting the association of talcum powder use with increased risk of ovarian cancer* (2019) (unpublished manuscript)) ("Manuscript") at 2.

[36] Saed Rep. at 16.

[37] Saed Dep. Vol. II 464:2-11.

[38] Saed Dep. Vol. I 268:4-269:4.

[39] *Id.* 268:25-269:4.

[40] Saed Rep. at 17.

[41] Saed Dep. Vol. I 265:10-15.

initial reaction of cells to particulate exposure. It is unreasonable to extrapolate from these findings that cells are therefore "oncogenic" and any observed fluctuations in proliferation and apoptosis are permanent. Dr. Saed's findings on proliferation and apoptosis do not seem to have any bearing on whether talc increases the risk of ovarian cancer.

C. Limitations of Results and the Need for Further Study

Alterations in Expression Levels and Activities of the Enzymes Studied Do Not Equate to an Altered State of Oxidative Stress in the Cultured Cells: As described in much of the evidence submitted by Dr. Saed in the context of expert testimony, including laboratory notebooks, the transcript of his deposition, and perhaps most succinctly, the manuscript by Fletcher *et al*. summarizing his findings, he consistently states and otherwise implies, many times, that decreased expression and activity of the antioxidant enzymes CAT and SOD3, increased expression and activity of the pro-oxidants iNOS, NO2-/NO3-, and MPO, and decreased expression and activity of antioxidant enzymes GSR and GPX "enhances the pro-oxidant state in . . . cells."[42] While he reports RNA levels ("expression") of these enzymes, as measured by qPCR, that are altered (up or down) following exposure to talc for 72 hours, he frequently conflates "expression and activity" of these enzymes as assessed by an ELISA, which measures protein levels.[43] The reactions that these enzymes catalyze may alter the levels of reactive oxygen species (typically nitrogen- or oxygen-based), but these reactive oxygen species are very unstable and cannot be measured by an ELISA. As best as I can tell from his laboratory notebooks, and from the content of the manuscript, he is using protein levels, as measured by an ELISA, to estimate the amount of enzymatic activity that a certain quantity of protein may have. This is an indirect and misleading presentation of the data. *Regardless*, none of these data are indicative of an increased pro-oxidant state in the cultured cells *in vitro*, much less *in vivo*.

The Single Nucleotide Polymorphism (SNP) Findings are Vague and of Questionable Relevance: ***First***, Dr. Saed has not established that his findings actually represent mutations, as he claims in his manuscript. In Table 2, he lists what he believes to be talc-induced genetic mutations resulting in SNP genotype switches in "key redox enzymes."[44] But as he acknowledged at his deposition, he was not "able to estimate the volume of cells that this genotype switch occurred in."[45] Rather, his technique only reports whether there is a "population of cells that acquired th[e] genotype" at issue.[46] This limitation is significant because it cannot rule out the possibility that the cells under treatment had one of three possible SNP genotypes (heterozygous, homozygous for minor allele, or homozygous for major allele) already, prior to treatment – in other words, that Dr. Saed was not finding treatment-induced mutations at all, but

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[42] Manuscript at 2.

[43] *Id.* at 20-22 (panels A and B of each figure show RNA expression, while panels C and D of each figure show protein levels as measured by ELISA).

[44] *Id.* at 19 (Table 2).

[45] Saed Dep. Vol. I 198:13-199:15.

[46] *Id.*

rather preexisting genetic variability that became manifest after the expansion of one or another subpopulation of cells in culture as a result of variable proliferation of a heterogeneous cell population. Indeed, the term "single nucleotide polymorphism" is by definition a type of genetic variation that exists in a population at a particular nucleotide position in a particular gene. In other words, polymorphisms represent naturally occurring genetic variants, not "mutations", at least in the context of putative carcinogen-induced mutagenesis over a 72-hour period. This occurs when a specific nucleotide in a specific gene is variable throughout a population, occurring when one genetic variant is inherited from one parent and the other genetic variant is inherited from the other parent. At a typical SNP site in the human genome, an individual may be homozygous for the SNP (for example T/T or C/C), or heterozygous for the SNP (C/T). These are not mutations. They represent the genetic basis of human phenotypic variation, and one may find SNPs in the great majority of human genes. This well-established genetic phenomenon throws Saed's entire claim of talc-induced mutations into doubt.

Second, none of the SNPs identified by Dr. Saed in his background discussion of ovarian cancer-associated polymorphisms was observed in his talc study. Dr. Saed broadly states in his report that SNPs in genes that code for certain enzymes (such as *CAT*, *GPX1*, *GSR* and *SOD2*) have been associated with increased ovarian cancer risk.[47] In making this statement, Dr. Saed relies, in part, on the Belotte study, conducted in his lab, which actually found an association between a specific SNP in the *CAT* gene and ovarian cancer ***survival***, not risk. Dr. Saed fails to elaborate on his statement and only identifies three SNPs in redox genes that he claims are related to ovarian cancer risk: rs1001179 (reducing CAT activity), rs4673 (reducing CYBA activity) and rs2333227 (occurring in the *MPO* gene).[48] The rs1001179 polymorphism is actually associated with ovarian cancer survival, not risk.[49] And a meta-analysis of 43 case-control studies involving various types of cancer found no association between the rs2333227 polymorphism (*MPO*) and an increased cancer risk.[50] Regardless, none of the underlying studies referenced by Dr. Saed is a genome-wide association study (GWAS) that examined the prevalence of a given SNP in a larger population of ovarian cancer patients. In other words, even if these three SNPs were hypothesized to be associated with ovarian cancer risk in isolated, statistically-underpowered investigations, their significance when it comes to the broader questions of ovarian cancer risk in the general population has not been established.

Perhaps recognizing this gap in his analysis, Dr. Saed also lists a number of additional SNPs identified by GWAS that influence ovarian cancer risk.[51] It is unclear whether these polymorphic variants are associated with an increased or decreased risk. None of the variants

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47 Saed Rep. at 7-8.

48 *Id.* at 8.

49 Belotte J et al., *A single nucleotide polymorphism in catalase is strongly associated with ovarian cancer survival.* PLoS One. (2015) 24:10(8):e0135739.

50 Chu H et al., *The* MPO *−463G>A polymorphism and cancer risk: a meta-analysis based on 43 case–control studies*. Mutagenesis. (2010) 25(4):389-95.

51 Saed Rep. at 8.

seem to occur in protein-coding regions except possibly rs2072590, which is "located at 2q31" within "a family of *HOX* genes."[52] The remaining variants occur "near" *BNC2* and *MERIT40*, "downstream" of *MYC*, and "intronic" to *SKAP1* and *TIPARP*.[53] At most, these SNPs could theoretically function to regulate the expression of genes, but not functions of the encoded protein, if they have any effect at all. It is certainly far from evident that any of these genes is involved in the redox state of cells.

Third, none of the "mutations" that Dr. Saed observed in his talc-treated cells has been reported by GWAS to be associated with an increased ovarian cancer risk. It should be noted that many SNPs are "silent," in that they do not result in any change in activity by the protein, and Dr. Saed has failed to show that the SNPs he claims resulted from talc-induced genotype switching are related to altered functions of the genes under study. Dr. Saed lists *CAT* (rs769217), *NOS2* (rs2297518), *GSR* (rs2448), *GPX1* (rs2448) and *SOD3* (rs2536512) genetic variations in Table 2 of his manuscript.[54] He was unable to state whether these SNPs have been reported to occur in women using talc.[55] And as discussed below, the observed "mutations" in *CAT*, *NOS2*, and *GPX1* fail to support his conclusions, for a number of additional reasons. Notably, the *GSR* and *SOD3* genes were not affected at all by talc treatment, as reported in Table 2.

CAT (rs769217) SNP. Dr. Saed did not observe this "mutation" in A2780 and SK-OV-3 cell lines. If this mutation is the mechanism by which talc allegedly increases ovarian cancer risk, it is unclear why the mutation is not commonly seen across all talc-treated cells. Dr. Saed makes many logical leaps to connect this genetic variant to an elevated cancer risk.

First, Dr. Saed states that the SNP results in an isoleucine to threonine amino acid change, but no more information is provided as to how or whether this change affects protein function.[56] Does the mutation alter the catalytic site of the enzyme? Does it affect secondary and tertiary structures of the protein or modify its interactions with other molecules? Dr. Saed's only observation is that talc-treated cells exhibit decreased *CAT* expression and catalase activity. However, he acknowledges in his report that these changes may be caused by other mutations in *CAT*, and not the rs769217 variant itself.[57] In fact, it would be much more logical to conclude that lower amounts of CAT protein in a cell would result in lower CAT activity (converting hydrogen peroxide to water and oxygen). Nevertheless, there are many straight-forward follow-up experiments that Dr. Saed could have conducted to understand the specific effect of the rs769217 genetic variant on catalase activity (if any). Scientists regularly create cell lines with targeted mutations through the use of genetic editing tools (such as CRISPR/Cas9), to study the impact of specific genetic mutations on protein functions. Dr. Saed could have repeated his

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52 *Id.*

53 *Id.*

54 Manuscript at 19 (Table 2).

55 Saed Dep. Vol. I 225:17-226:3.

56 Manuscript at 19.

57 Saed Rep. at 18.

ELISA assays and done pull-downs of the catalase protein in normal cells and cells with targeted mutations to understand whether and how the rs769217 mutation affected the catalase function and its interaction with other molecules (including its function as a tetramer). Only with these sorts of follow-up experiments could Dr. Saed actually attribute a causal relationship between this specific genetic variant and the protein activity observed.

The minor allelic frequency (MAF) of the rs769217 SNP was described as 12.3%.[58] As presented, this figure can only be derived from the genotypes of large numbers of individuals in a population. For a single individual, the MAF would by necessity be 0, 50%, or 100%. These are basic principles of human genetics. In the talc treatment experiments, data are presented as Allele 1 and Allele 2 scores with and without talc treatment; in the case of TOV-112D cells, for example, the C/C genotype at rs769217 becomes C/T following talc treatment with scores of Allele Amp Scores of 0.67 and 0.88.[59] Although it is not clear exactly what these scores represent (the total is greater than 1.0), it may be assumed that a substantial proportion of the cells exposed to a dose of talc for 72 hours sustained a C to T mutation. I have never witnessed such potent mutagenesis by any agent – especially within a narrow 72-hour post-treatment window. Dr. Saed was similarly unable to recall any agent that has produced such rapid, robust mutagenesis.[60] It is highly unlikely that the increased MAF is due to genotoxicity that is unique to talc, considering a previous study found that talc was not genotoxic.[61] Rather, the high MAF is likely the result of general genotoxicity associated with the introduction of extremely high dosages of foreign particulate into cell cultures, the selective expansion of small numbers of cells present in culture with the MAF, otherwise undetectable, as the cells were induced to proliferate by talc exposure, some sort of experimental error, or all of the above. The inclusion of appropriate control experiments (as previously described) could have shed light on these questions. Finally, as noted elsewhere in this report, the allele frequencies for all the studied SNPs should have been presented in a quantitative fashion, rather than qualitative. For a mutation to be "fixed" in an affected cell, the cell must obviously undergo division to two daughter cells. That specific SNP sites that happened to be associated with enzyme activity of the "critical" genes under study underwent qualitative mutagenesis from one nucleotide to another in 100% of the talc-treated cells, in 72 hours, is not only implausible, it is ***impossible***, in light of the doubling time of proliferating cells.

SOD3 (rs2536512) and *GSR* (rs8190955) mutations. Dr. Saed's report states that these "SNP genotypes were not detected in any cell line."[62] Part B of Table 2 confirms that neither the control nor talc-treated cell lines had mutations at these locations.[63] However, the first part of

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58 Saed Dep. Vol. I, Ex. 1 at SAED000078.

59 *Id.* at SAED000080.

60 Saed Dep. Vol. I 252:3-7.

61 Endo-Capron S et al., In vitro *response of rat pleural mesothelial cells to talc samples in genotoxicity assays (sister chromatid exchanges and DNA repair).* Toxicol In Vitro. (1993) 7(1):7-14.

62 Saed Rep. at 18; Manuscript at 11.

63 Manuscript at 19.

the table still lists the MAF of mutations as 19.1% and 47.6%, respectively.[64] As for the CAT gene data above, it is unclear from whence the MAF data are derived. Is it a calculation of allelic frequency based on the total pooled alleles from all of the talc-treated cells? Is it an average of the MAF values calculated individually for each of the talc-treated cell lines? Is it the naturally-occurring frequency of the mutation in the general population? If it does refer to the frequency in the general population, what proportion of cells treated with talc actually displayed these mutations?

Regardless of how the MAF data were calculated, if no SNP genotypes were detected in the cell lines, how can these *SOD3* and *GSR* mutations still be attributed to changes in redox activity and provide any basis for Dr. Saed's theory that talc exposure leads to mutations associated with an increase in ovarian cancer risk?

NOS2 (rs2297518) mutation. The concerns described above also apply to the *NOS2* mutation. This mutation was not found in the talc-treated A2780 or TOV-112D cell lines, had a MAF of 17.3% and resulted in a serine to leucine amino acid change.[65] No additional studies were conducted to confirm that observed increases in protein activity were actually caused by the rs2297518 mutation.

GPX1 (rs3448) mutation. In addition to the concerns described above, other issues also undermine the significance of the *GPX1* findings. First, Dr. Saed focuses on the mutation because the "acquisition of chemoresistance by ovarian cancer cells is associated with a switch from *GPX1* SNP genotype to the normal *GPX1* genotype."[66] It is unclear how any chemoresistance finding in already cancerous cells is relevant to understanding whether an association exists between talc exposure and ovarian cancer risk. Among genes coding for glutathione peroxidase enzymes, only the rs6456822 SNP in *GPX6* has been reported as having a genome-wide significance for association with serous epithelial ovarian cancer risk.[67] Simply put, Dr. Saed does not provide any basis for why the rs3448 genetic variant is associated with ovarian cancer risk.

Dr. Saed did not observe the *GPX1* conversion in one of the normal cell lines (HOSEpiC) after exposure to talc. As with the *CAT* mutation, if this mutation is the mechanism by which talc allegedly increases ovarian cancer risk, it is unclear why the mutation did not occur in all normal cells treated with talc. Showing this mutation occurs in all normal cells treated with talc would be the first step toward understanding any biological mechanism whereby talc allegedly leads to an increased risk of ovarian cancer.

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64 *Id.*

65 *Id.*

66 Saed Rep. at 19.

67 Kuchenbaecker KB et al., *Identification of six new susceptibility loci for invasive epithelial ovarian cancer.* Nat Genet. (2015) 47(2):164-71.

Finally, Dr. Saed describes the amino acid changes and effect on protein activity for the *GPX1* mutation as "unknown."[68] Dr. Saed has no idea why the mutation is significant to his opinion on talc and ovarian cancer risk other than the fact that the mutation occurs in a gene involved in redox activity. The mere existence or creation of a mutation is not necessarily biologically significant. For example, the SNP could be a synonymous mutation that does not result in any amino acid change in the resulting protein and has no consequence on glutathione peroxidase enzyme function. If the SNP did result in an amino acid change, the change could be inconsequential because it does not affect the activity of the enzyme, the secondary or tertiary structures of the protein or how the protein interacts with other molecules. As it stands, there is no basis for the relevance of the *GPX1* mutation in studying ovarian cancer risk.

My interpretation of the experimental design and presentation of data related to the measurement of SNP genotypes in several genes involved in the general oxidative state of the cell, after exposure to talc, is that Dr. Saed has conflated mutagenesis with normal genetic variation, especially as the latter may exist in a highly heterogeneous state in cells cultured *in vitro*. It is not at all clear how these data bear on the purported risk of talc for the development of ovarian cancer. This view would seem to be shared by Reviewer #1 of the manuscript submitted to *Gynecologic Oncology*, who writes, "The significance of SNP alterations should be further clarified."[69]

If Dr. Saed had been interested in demonstrating that talc was indeed mutagenic (creating mutations) in his cell lines, the most appropriate experiments would have examined global mutagenesis in a much broader context. One potential experiment would involve comparing talc-treated cells to untreated cells with respect to potential mutations generated throughout the entire exome (coding region of the genome). This experiment would have involved extraction of DNA from treated vs. untreated cells, followed by sequencing of the entire exomes of these cells using next-generation DNA sequencing technology. This technology is typically available in core facilities of most research universities and academic medical/cancer centers, and if not, is readily performed by myriad commercial laboratories for a modest cost. An alternative approach would have been to perform next-generation DNA sequencing analysis of a panel of several hundred genes known to be involved ("driver genes") in carcinogenesis when mutated. Such analyses are also performed by many commercial laboratories.

In summarizing my conclusions on scientific clarity and relevance of the SNP studies, I can only conclude that the rationale of studying talc-induced mutagenesis occurring ***exclusively*** at SNP sites in some of the genes encoding enzymes under study, including the anti-oxidant enzymes CAT, GSR, GPX1, and SOD3, and the pro-oxidant enzyme NOS2, appears to represent a chain of logic by Dr. Saed that would correlate talc-induced mutations at these specific sites with altered enzymatic activity of the encoded proteins, followed by increased oxidative stress in the affected cells; this complex theoretical sequence of talc-induced events in cultured cells would appear to tie all of his various hypotheses together. Parenthetically, there is no evidence or

[68] Manuscript at 19.

[69] Gynecologic Oncology Decision at 2.

suggestion provided in Dr. Saed's manuscript as to how the enzymes affected by talc exposure (expression levels) were so affected if they ***did not contain SNPs subject to mutagenesis*** and thus not studied at all (*MPO*), or ***did*** contain SNPs of purported functional consequence but ***did not sustain mutagenesis by talc*** (*GSR* and *SOD3*). These data are presented in Table 2 of Dr. Saed's manuscript. In my expert opinion, this experimental design and interpretation of results are deeply flawed, naïve, and the results regarding qualitative (as opposed to quantitative) mutagenesis at specific SNP sites are, candidly, very difficult to believe. I have expanded upon all the critical elements of this paragraph elsewhere throughout this Expert Report.

Limitations of Studies *in vitro*: Even if Dr. Saed's research methodology were flawless, and his conclusions unassailable, his studies *in vitro* would not establish a mechanism of carcinogenesis *in vivo*. The most even Dr. Saed claims to have actually shown with his experiment is a change in the levels of RNAs and proteins that encode certain proteins, changes in the activities of some of these proteins (by inference), an increase in cell proliferation and a decrease in apoptosis in response to talc exposure; but there is an enormous gap between such findings in a petri dish and proving that a particular agent is actually a probable cause of ovarian cancer.

Indeed, as a general rule, a study *in vitro* cannot, by itself, support conclusions about anything that happens in actual animal or human tissues. At most, careful studies *in vitro* may generate hypotheses that may be tested with follow-up studies using models *in vivo*, e.g., animals. The comments on Dr. Saed's manuscript reflect this principle. According to Dr. Saed's deposition testimony, *Gynecologic Oncology*[70] declined to publish his paper, and a reviewer explained that he "needed to do *in vivo* . . . animal experiments."[71] I note, too, that Dr. Saed volunteered at his own deposition that, in order to determine whether his experiments truly emulated chronic inflammation in humans, he would "have to do animal studies."[72]

The need for studies *in vivo* to evaluate Dr. Saed's results *in vitro* is especially glaring here, because previous work *in vivo* on the relationship between talc and ovarian cancer tends to refute, rather than support, Dr. Saed's conclusions. I am not aware of any research *in vivo* specifically addressing the effects of talcum powder exposure on oxidant and anti-oxidant enzymes and resultant oxidative stress in human cells. Two animal studies, however, have shown no increase in ovarian cancer development following talcum powder treatment. Hamilton, *et al.*, injected rats with mega-doses of talc adjacent to the ovaries, and reported no inflammation or neoplasia.[73] Keskin, *et al.*, exposed rats to talc either intra-vaginally or on the perineum. While certain infections developed (likely because the talc was not sterile), there was

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[70] Dr. Saed testified that he submitted his manuscript to a journal called "*OB-GYN Oncology*." I am aware of no journal with that name, and subsequent document productions from Dr. Saed make clear that he intended to refer to *Gynecologic Oncology*.

[71] Saed Dep. Vol. I 46:22-47:2; *see also* Gynecologic Oncology Decision.

[72] Saed Dep. Vol. II 542:16-25.

[73] Hamilton TC et al., *Effects of talc on the rat ovary*. Br J Exp Pathol. (1984) 65(1):101-6.

no neoplastic change in any of the exposed animals.[74] Dr. Saed is capable of performing studies *in vivo* to challenge these conclusions, but said at his deposition that he lacks the time and the money for it.[75] In light of the data from earlier studies, I am skeptical that Dr. Saed's findings could be replicated *in vivo*, and without such replication, they are insufficient to reliably suggest the carcinogenic mechanism that he proposes.

Relatedly, Dr. Saed is presupposing that talc can travel to the fallopian tubes or ovaries and cause inflammation there, but his *in vitro* experiments obviously cannot evaluate that assumption, and support from existing research is lacking. In fact, Dr. Saed's suggestion that it is widely accepted that talc applied to a woman's underwear will travel to her ovaries against gravity[76] and that studies of sperm are somehow relevant to this question[77] ignores fundamental anatomy. Notably, the often-cited study regarding the presence of talc in ovarian tissue of women with ovarian cancer discovered talc both in women who reported perineal talc use and women who did not, suggesting that the talc came from a different source.[78]

With respect to Dr. Saed's assertion that his data support a role for oxidative stress (presumably produced by talc exposure) in ovarian carcinogenesis, in addition to my concerns raised in this report, both Reviewers for *Gynecologic Oncology* commented on this assertion specifically as it was articulated in Dr. Saed's manuscript.[79] Reviewer #1 writes, "The first bulleted highlight [the Journal requires a list of bulleted highlights of research papers submitted for publication], 'Oxidative stress is a key mechanism to the initiation and progression of ovarian cancer' is not supported by this investigation and should be omitted."[80] Reviewer #2 writes, "While changes in redox potential play an important role in in tumor biology in general, the present data are insufficient to back up the claim that talcum is central to the development of ovarian cancer."[81]

Finally, Dr. Saed appears to take for granted that ovarian cancer is caused by inflammation, but this, too, has not been established. Dr. Saed essentially ignores the body of science suggesting that chronic inflammation does not play a role in the development of ovarian cancer,[82] as well as

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[74] Keskin N et al., *Does long-term talc exposure have a carcinogenic effect on the female genital system of rats? An experimental pilot study*. Arch Gynecol Obstet. (2009) 280(6):925-31.

[75] Saed Dep. Vol. I 50:10-13.

[76] Manuscript at 8.

[77] *Id.* (citing Kunz G et al., *The uterine peristaltic pump. Normal and impeded sperm transport within the female genital tract*. Adv Exp Med Biol. (1997) 424:267-77; Leyendecker G et al., *Uterine peristaltic activity and the development of endometriosis*. Ann NY Acad Sci. (2004) 1034:338-55; Zervomanolakis I et al., *Physiology of upward transport in the human female genital tract*. Ann NY Acad Sci. (2007) 1101:1-20

[78] Heller et al., *The relationship between perineal cosmetic talc usage and ovarian talc particle burden.* Am J Obstet Gynecol. (1996) 174(5):1507-10.

[79] Gynecologic Oncology Decision at 2-3.

[80] *Id.* at 2.

[81] *Id.*

[82] Malmberg K et al., *Serous tubal intraepithelial carcinoma, chronic fallopian tube injury, and serous carcinoma development*. Virchows Arch. (2016) 468(6):707-13; Rasmussen et al., *Pelvic inflammatory disease and the risk* *(cont'd)*

studies that considered whether aspirin use and anti-inflammatory drugs reduced the risk of ovarian cancer,[83] with mixed results. As the Malmberg study concluded after finding no significant correlation between histological signs of inflammation and serous ovarian cancer, "Additional studies are needed to further evaluate the role of inflammation in carcinogenesis in the fallopian tube and its clinical implications of preventing serous carcinoma."[84]

Need for Further Study: In addition to the concerns noted above regarding the limitations of the studies performed *in vitro*, and the inappropriate conclusions drawn from them, several related types of studies were notably ***not*** performed by Dr. Saed in the context of providing evidence central to the fundamental assertion of plaintiffs that perineal talc use causes ovarian cancer. It is widely accepted in the cancer research community that there are several relatively straightforward assays that may be used to support the hypothesis that "normal" cells cultured *in vitro* have been stimulated by some type of exposure or manipulation (talc treatment in this case) to progress toward, or to fully develop, a neoplastic phenotype. These assays include, but are not limited to, the assessment of loss of contact inhibition by cells cultured in a petri dish *in vitro*, the acquisition of anchorage independent growth potential (as assessed by culturing cells in suspension in soft agar), and perhaps the most compelling experiment, demonstrating that the treated cells have obtained neoplastic potential as assessed by their ability to form tumors following subcutaneous injection into athymic ("nude") mice. All these assays employ standard, well-established methodologies, and could have been readily performed by Dr. Saed using the "normal" cell lines described in his studies. As discussed earlier, none of these studies could have been performed using the three ovarian carcinoma cell lines described, however, since they have already undergone neoplastic transformation (in the humans from whence these cancers arose, and from whence the cell lines were derived). Notably, the three ovarian carcinoma cell lines could have been used as positive controls for the three assays described above, as they would have certainly demonstrated loss of contact inhibition in a petri dish, anchorage independent growth in soft agar, and tumorigenicity in athymic mice. I note that Dr. Saed himself proposed to do the second assay just mentioned involving suspension in soft agar, even stating in his proposal that actually demonstrating "neoplastic transformation" would be "critical in establishing a cause and effect relationship" between talc exposure and ovarian cancer,[85] but as he confirmed at his deposition, he never performed such a study.[86]

(cont'd from previous page)
of ovarian cancer and borderline ovarian tumors: A pooled analysis of 13 case-control studies. Am J Epidemiol. (2017) 185(1): 8–20; Zhou et al., *Pelvic inflammatory disease and the risk of ovarian cancer: a meta-analysis*. Cancer Causes Control. (2017) 28(5):415-28.

[83] Ni X et al., *Meta-analysis on the association between non-steroidal anti-inflammatory drug use and ovarian cancer*. Br J Clin Pharmacol. (Jan. 2013) 75(1):26-35.

[84] Malmberg et al. (2016) at 712.

[85] Saed Dep. Vol. II, Ex. 44 at 3, The Role of Talc Powder Exposure in Ovarian Cancer: A Mechanistic Approach.

[86] Saed Dep. Vol. II 513:6-14.

D. Concerns Regarding Data and Handling/Manipulation of Laboratory Notebooks Generally

I have carefully studied three PDF files (in color) representing scanned portions of laboratory notebooks pertaining to the studies discussed in this Expert Report, that were provided by Dr. Saed, as well as Dr. Saed's deposition testimony about the conduct of his studies. My understanding is that the three PDF files accurately reflect the contents of some portion of the laboratory notebooks related to the studies discussed herein, and that the content of the notebooks was produced by Dr. Saed or members of the Saed laboratory working under his supervision. As a result of miscalculations, changing of dates on particular pages, whiting-out of data or notes, addition of data or notes to certain pages on different dates, the taping of data sheets cut from another source over data or notes previously existing on certain pages, the presence of data and other information in these notebooks that contradict Dr. Saed's statements during deposition as well as data and conclusions reached in the manuscript describing these studies that were submitted to at least two biomedical journals, and other irregularities too numerous to describe in detail, I have reached the following conclusions: 1) Some of the data and handwritten notes in these notebooks were intentionally manipulated; 2) Some of the data in these notebooks were selectively excluded from the final conclusions ultimately manifest in the manuscript submitted for publication; 3) Some of the data in these notebooks and conclusions drawn from them are internally inconsistent; 4) The handling of these laboratory notebooks and the recording of data and notes therein are egregiously inconsistent with the very minimum of well-accepted standard operating procedures with respect to the handling of laboratory notebooks and the recording of data and notes in the context of laboratory research; and, ***5) It is my expert (as defined on pages 2 and 3 of this Report) opinion that some of the data in these notebooks are at the very least unreliable, and at worst fabricated, and that the conclusions drawn from these data, as a whole, are thus unbelievable and essentially worthless with respect to the written and stated claims relating to a possible mechanism(s) through which talc may induce tumorigenesis in cultured cells specifically, and by multiple layers of illogical extension, through which talc may induce ovarian cancer in women exposed to talc generally.***

For the record, I received three notebook files. The first ("Expert Report Notebook Files") was described as the laboratory notebook that relates to Dr. Saed's work for his expert report. It consists of 97 pages (with what would appear to be printed stickers in the bottom corner of each page labeled SAED000001(color) – SAED000097(color)). There are handwritten numbers on the bottom corner of each page, beginning with "30" on page 1 and "124" on page 97. There are two un-numbered pages inserted between the handwritten pages 33 and 35, and one un-numbered page inserted between the handwritten pages 39 and 40, possibly accounting for the discrepancy of two "missing" pages with respect to the handwritten numbered version. For orientation, page 1 (or 30) contains color photographs of the front and back of a commercial container of "Johnson's baby powder."

The second laboratory notebook file ("Abstract Lab Notebook Files") contains a table of contents on the un-numbered first page, with a series of dates, 9/26/2017 – 10/20/2017, descending vertically on the left side, and page numbers from 38-63 descending vertically on the right side. The pages are hand-numbered in the bottom corner, beginning with 38 after the TOC

page and ending with 61, prior to the last page consisting of a scientific poster prepared for presentation.

The third laboratory notebook file ("Preliminary Work Notebook Files") represents the first 30 pages that are missing from the Expert Report Notebook Files. My understanding is that plaintiffs did not originally share it with defendants because they characterized it as containing only preliminary work.[87] It begins with a table of contents on the un-numbered first page, with a series of dates, 10/15/2017 – 10/6/2017, descending vertically on the left side, and page numbers from 1-124 descending vertically on the right side. Pages 25-30 are missing from the table of contents. The pages are hand-numbered in the bottom corner, beginning with 1 after the TOC page containing a photograph of a container of "Talc" from Fisher Chemical. The next page is un-numbered and contains the same color photographs of a commercial container of "Johnson's baby powder" that appeared in the Expert Report Notebook Files. The next page is numbered 2 and the rest are numbered consecutively 3-24.

Examples of some of the irregularities described in the first paragraph of section IV.D of this Expert Report (above) include:

1) Pages from another source taped onto the laboratory notebook page, white-out present in both files, including dates whited out and single entries that are made with ink of a different color than the text otherwise filling the same page. I further note that apparent manipulation of the dates has resulted not only in lab books that have entries out of chronological order, but also statements that cannot possibly be true. For example, page 25 of the Expert Report Notebook Files is dated January 7, 2018, and claims to be recording protein extractions from samples 356 to 386.[88] The first line after the top of this page states that the cells were seeded on January 3, 2018.[89] The very next page identifies samples 356 through 386.[90] But exactly the same samples are also identified on page 20 of the Preliminary Work Notebook Files (which, as I note above, plaintiffs initially withheld from production on the ground that it was unrelated work). *That* page refers to the actual seeding of the samples and is dated *February 1, 2018* – or *nearly a month after* protein extractions were supposedly taken from the same samples (which had not been created yet).[91] There is no question that these pages in the separate parts of the Notebooks are referring to the same samples – Dr. Saed said so himself at his deposition, calling the samples "exactly the same."[92] In fact, the February 1 date in the Preliminary Work Notebook Files follows a "1/3" date that has been crossed out[93] – a date that matched the date referred to on page

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87 Saed Dep. Vol. I 13:18-14:10, 15:24-16:1.

88 Saed Dep. Vol. I, Ex. 1 at SAED000025(color).

89 *Id.*

90 *Id.* at SAED000026(color).

91 Saed Dep. Vol. II, Ex. 23 at Ghassan Saed's Talc Study Lab Notebook – Preliminary Study ("Preliminary Work Notebook Files") at 20.

92 Saed Dep. Vol. II 390:7-17.

93 Preliminary Work Notebook Files at 20.

25 of the Expert Report Notebook Files[94] as the date when the cells were supposedly seeded. These changes suggest that the dates were intentionally manipulated (rather than, for example, that the author mistakenly believed that it was January 3 on February 1).

2) Throughout the Preliminary Work Notebook Files, the handwritten page numbers are invariably smudged, suggesting either erasure and writing over, or white-out and writing over.

3) On page 19 of the Preliminary Work Notebook Files, there is a handwritten entry as follows: "1/31/18 – The presence of 1000 µg/ml is physically killing the cells. – We need to decrease dose."[95] In none of the pages preceding page 19 of the Preliminary Work Notebook Files, or in any section of the Abstract Lab Notebook Files (containing experiments ostensibly performed prior to 1/31/18), is there evidence of such toxicity. In fact, data related to gene expression (as assessed by RNA levels) are readily obtained at doses of 20, 100 and 1000 µg/ml. In some cases, gene expression of particular enzymes is higher at 1000 µg/ml than at 20 or 100 µg/ml, inconsistent with cells being "physically killed" at 1000 µg/ml. In addition, the amount of RNA obtained from a given number of cells is similar in control vs. treated cells, and from cells treated at various doses (20 – 1000 µg/ml). These data are also inconsistent with a greater proportion of "dead" cells at 1000 µg/ml. What is ***clearly*** apparent, however, is that gene expression and CA-125 secretion levels at a dose of 1000 µg/ml do not follow a traditional "dose-response" (a biological response becoming increasingly higher or lower in response to an increasing dose of test substance). In quantitating CA-125 secretion, for example, sometimes the amount does not change with talc, sometimes it is lower with talc, and sometimes it is higher with talc, compared to DMSO control treatment of the same cells.[96] This phenomenon does not fit with a central tenet of Dr. Saed's conclusion, which is that there is a clear dose-dependent response in terms of gene expression, protein "activity," CA-125 secretion, etc., following talc exposure. This selective exclusion of data in order to fit data to a particular hypothesis or conclusion, "cherry-picking" data to use a colloquialism, is unsound scientific methodology of the highest order.

4) With respect to data points themselves, there is clear evidence of error (human or machine) in terms of simple arithmetic calculations. For example, in a random spot check (by me) of raw data in the Expert Report Lab Notebook Files, consider the computer-generated table (whether populated by a human or a machine being impossible to know) on page SAED000033(color). These data relate to an ELISA-based measurement of catalase "protein/activity" following exposure of cultured cells to talc at doses of only 5, 20 and 100 µg, (presumably per ml?) and the table is dated 1/11/18.[97] This date is 20 days before 1/31/18, the date upon which, in the

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94 Saed Dep. Vol. I, Ex. 1 at SAED000025(color).

95 Preliminary Work Notebook Files at 19.

96 For example, see Preliminary Work Notebook Files at 13.

97 Saed Dep. Vol. I, Ex. 1 at SAED000033(color).

Preliminary Work Notebook Files, a notation is found that, "The presence of 1000 µg/ml is physically killing the cells…"[98]

Regardless, if one considers the data table in question, the first horizontal row concludes on the far right with an "Average" value of 11.07 for three replicate values of 9.98, 11.63, and 10.50.[99] The correct average would have been 10.70. In horizontal line two of the same table, the "Average" value is listed as 9.13 for three replicate values of 9.18, 10.64, and 9.09.[100] The correct average would have been 9.64. Thus, the recorded difference between "control" A2780 cells and talc-treated (5 µg) A2780 cells is 1.94 nmol/min/ml[101]; the actual difference is 1.06 nmol/min/ml, a much smaller difference. A "larger difference" in this case would have been more consistent with the experimental hypothesis and conclusions, which of course could be simply coincidental, the arithmetic errors notwithstanding. There are other examples of these kinds of data errors throughout Dr. Saed's work, several of which were covered at his second deposition.[102]

5) I have also reviewed multiple drafts of Dr. Saed's manuscript, including the version of it that was rejected by *Gynecologic Oncology* and the version later accepted by *Reproductive Sciences*. Of particular interest is the fact that the earlier submission to *Gynecologic Oncology* claimed to have observed effects of talc after only 48 hours of treatment – a fact directly addressed by one of the reviewers in the rejection letter, who wrote that the "fact that SNPs were changed following such short exposure to talcum is surprising and makes one wonder what the biological effect of such changes might be."[103] Curiously, Dr. Saed's subsequent submission to *Reproductive Sciences* changed the stated time of treatment to 72 hours – but includes many of the same tables that were included in the submission to *Gynecologic Oncology*, with exactly the same data for each dose of treatment, but with the exposure period changed from 48 hours to 72 hours. And Dr. Saed's report states that he treated talc "for 48 hours"[104] – a discrepancy from his latest manuscript that he attempted to explain as "a typo" in his report at his deposition.[105] Of course, another possibility is that Dr. Saed decided that 72 hours of treatment would appear more credible and that he simply revised this reference in his manuscript without rerunning the experiments before he resubmitted but forgot to make the same change to his report.

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98 Preliminary Work Notebook Files at 19.

99 Saed Dep. Vol. I, Ex. 1 at SAED000033(color).

100 *Id.*

101 *Id.* at SAED000090(color).

102 *See, e.g.*, Saed Dep. Vol. II 450:24-452:6, 452:22-453:24 (additional averaging errors).

103 Gynecologic Oncology Decision at 2.

104 Saed Rep. at 14.

105 Saed Dep. Vol. I 185:6-186:7.

E. Additional Concern

Improper financial disclosure: Dr. Saed's insufficient conflict-of-interest disclosure violates publishing principles and further indicates that his opinions are not reliable. Although there is no single definitive standard for an appropriate conflict-of-interest disclosure, failures to disclose conflicts of interest have undermined the faith of both the public and healthcare professionals in the quality of scientific and medical literature.[106] As such, most reputable journals have developed their own conflict-of-interest disclosure policies, and various voluntary organizations have advanced model standards that function as persuasive guidelines. Dr. Saed's minimal disclosure violates both these model policies and the policy in place at *Reproductive Sciences*,[107] the journal in which his manuscript is to be published.

For example, the International Committee of Medical Journal Editors states that authors should disclose "all financial or personal relationships that might bias or be seen to bias their work" and, in particular, notes "[f]inancial relationships (such as . . . paid expert testimony)" as the most obvious type of conflict of interest.[108] The World Association of Medical Editors has set forth a similar policy.[109] In keeping with these principles, *Reproductive Sciences* requires all authors to make a "specific" declaration of "any financial relationship" that the author has and the "interests" of the sponsoring organization, and to include any information "that might represent an appearance of a conflict of interest" in the cover letter.[110] Dr. Saed admits that he did not include any such information in his cover letter.[111] Dr. Saed did acknowledge elsewhere that he "acted as a consultant regarding this topic for a fee."[112] He did not link his consultancy to his manuscript in any way, much less disclose that plaintiffs' counsel funded the specific study that he submitted. Nor did he disclose that he functioned as more than a consultant, but as a testifying expert witness. Indeed, he did not even disclose for whom he consulted – whether it was a party, such as plaintiffs' counsel, with an interest in showing talc to be dangerous, a party, such as an industry player, with an interest in showing talc to be safe, or an unbiased organization. Therefore, reviewers, and ultimately readers, could not evaluate his conclusions with appropriate context in mind.

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106 Blum JA et al., *Requirements and definitions in conflict of interest policies of medical journals.* JAMA. (2009) 302(20):2230-4.

107 *See* Saed Dep. Vol. I, Ex. 12 at 3 (Sage Publishing Reproductive Sciences Webpage); *see also* Sage Publications, Declaration of Conflicting Interests Policy (2019), https://us.sagepub.com/en-us/nam/declaration-of-conflicting-interests-policy.

108 Int'l Committee Med. J. Editors, *Recommendations for the Conduct, Reporting, Editing, and Publication of Scholarly Work in Medical Journals* 3, http://www.icmje.org/icmje-recommendations.pdf (updated Dec. 2018).

109 *See* World Ass'n of Med. Editors, *Conflict of Interest in Peer-Reviewed Medical Journals*, http://wame.org/conflict-of-interest-in-peer-reviewed-medical-journals (updated July 25, 2009).

110 *See* Saed Dep. Vol. I, Ex. 12 at 3; *see also* Sage Publications, Declaration of Conflicting Interests Policy.

111 Saed Dep. Vol. I 156:10-19.

112 *Id.* 144:2-7; *see also id.* 142:1-2.

V. MATERIALS CONSIDERED

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2. Belotte J et al., *A single nucleotide polymorphism in catalase is strongly associated with ovarian cancer survival.* PLoS One. (2015) 24;10(8):e0135739
3. Blum JA et al., *Requirements and definitions in conflict of interest policies of medical journals.* JAMA. (2009) 302(20):2230-4
4. Chu H et al., *The* MPO *−463G>A polymorphism and cancer risk: a meta-analysis based on 43 case–control studies*. Mutagenesis. (2010) 25(4):389-95
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6. Deposition of Ghassan Saed, Ph.D., Vol. II, Feb. 14, 2019 (MDL No. 2738)
7. Didžiapetrienė J et al., *Significance of blood serum catalase activity and malondialdehyde level for survival prognosis of ovarian cancer patients*. Medicina (Kaunas) (2014) 50(4):204-8
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9. Expert Report of Daniel L. Clarke-Pearson, M.D. Nov. 16, 2018 (MDL No. 2738)
10. Expert Report of Ghassan Saed, M.D., Nov. 16, 2018 (MDL No. 2738)
11. Expert Report of Judith Wolf, M.D. Nov. 16, 2018 (MDL No. 2738)
12. Expert Report of Sarah Kane, M.D., Nov. 15, 2018 (MDL No. 2738)
13. Expert Report of Shawn Levy, Ph.D., Nov. 16, 2018 (MDL No. 2738)
14. Fletcher NM et al., LB-044 – Talcum Powder Enhances Cancer Antigen 125 Levels in Ovarian Cancer Cells and in Normal Ovarian Epithelial Cells (abstract) (2018) (Ex. 21 to Deposition of Ghassan Saed, Ph.D., Jan. 23, 2019 (MDL No. 2738))
15. Fletcher NM et al., Molecular basis supporting the association of talcum powder use with increased risk of ovarian cancer (2019) (unpublished manuscript) (Ex. 8 to Deposition of Ghassan Saed, Ph.D., Jan. 23, 2019 (MDL No. 2738))
16. Fletcher NM et al., *Specific point mutations in key redox enzymes are associated with chemoresistance in epithelial ovarian cancer*. Free Radic Biol Med. (2016) 102:122-32
17. Fletcher NM et al., Talcum Powder Enhances Oxidative Stress in Ovarian Cancer, Reproductive Sciences, Vol. 25, Suppl. 1, F-098 (abstract) (2018) (Ex. 20 to Deposition of Ghassan Saed, Ph.D., Jan. 23, 2019 (MDL No. 2738))
18. Forsberg L et al., *A common functional C-T substitution polymorphism in the promoter region of the human catalase gene influences transcription factor binding, reporter gene*

transcription and is correlated to blood catalase levels. Free Radic Biol Med. (2001) 30(5):500-5

19. Ghassan Saed's PCR EOC SRI Notebook (Ex. 9 to Deposition of Ghassan Saed, Ph.D., Jan. 23, 2019 (MDL No. 2738))
20. Ghassan Saed's Talc Study Lab Notebook – Preliminary Study (Ex. 23 to Deposition of Ghassan Saed, Ph.D., Feb. 14, 2019 (MDL No. 2738))
21. Gynecologic Oncology Email dated Sept. 19, 2018 re: GYN-18-1020: Final Decision (Ex. 35 to Deposition of Ghassan Saed, Ph.D., Feb. 14, 2019 (MDL No. 2738))
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25. Heller DS et al., *The relationship between perineal cosmetic talc usage and ovarian talc particle burden*. Am J Obstet Gynecol. (1996) 174(5):1507-10
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30. Keskin N et al., *Does long-term talc exposure have a carcinogenic effect on the female genital system of rats? An experimental pilot study*. Arch Gynecol Obstet. (2009) 280(6):925-31
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40. Rasmussen et al., *Pelvic inflammatory disease and the risk of ovarian cancer and borderline ovarian tumors: A pooled analysis of 13 case-control studies*. Am J Epidemiol. (2017) 185(1):8–20

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43. Scholler N & Urban N, *CA125 in ovarian cancer.* Biomark Med (2007) 1(4):513-23

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47. TOV-112D (ATCC® CRL-11731™), https://www.atcc.org/products/all/CRL-11731.aspx

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EXHIBIT A

Curriculum Vitae (02/04/19)

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505 Luenga Avenue
Coral Gables, FL 33146

Education:

Duke University, Durham, NC
B.S. (Psychology/Chemistry), 1980

NC State University, Raleigh, NC
M.S. (Toxicology/Biochemistry), 1982

NC State University, Raleigh, NC
Ph.D. (Toxicology/Biochemistry), 1986

Postdoctoral Training:

1986-1988: Environmental Pathology Fellowship
Department of Pathology
Lineberger Comprehensive Cancer Center
University of North Carolina School of Medicine
Chapel Hill, NC

1988-1990: Senior Staff Fellow
Cellular Carcinogenesis Section
Laboratory of Molecular Carcinogenesis
NIH/National Institute of Environmental Health Sciences
Research Triangle Park, NC

Positions and Appointments:

1990-1994: Head, Gynecologic Pathobiology Section
Laboratory of Molecular Carcinogenesis
NIH/National Institute of Environmental Health Sciences
Research Triangle Park, NC

1992-1994: Adjunct Assistant Professor (concurrent with primary position above)
Department of Epidemiology
University of North Carolina School of Public Health
Chapel Hill, NC

1994-1997: Associate Professor
Department of Obstetrics and Gynecology and Department of Genetics
Director, Gynecologic Oncology Research Laboratory
Member, Comprehensive Cancer Center
Member, Center for Research on Women's Health and Reproduction
Associate Member, Institute for Human Gene Therapy
University of Pennsylvania
Philadelphia, PA

1997-2003: Associate Attending Biologist
Gynecology Service, Department of Surgery
Clinical Genetics Service, Department of Medicine
Director, Gynecology and Breast Research Laboratory
Memorial Hospital for Cancer and Allied Diseases
Associate Member, Memorial Sloan-Kettering Cancer Center
New York, NY

2003-2006: Attending Biologist
Gynecology Service, Department of Surgery
Clinical Genetics Service, Department of Medicine
Director, Gynecology and Breast Research Laboratory (Department of Surgery)
Director, Diagnostic Molecular Genetics Laboratory (Department of Medicine)
Memorial Hospital for Cancer and Allied Diseases
Member (with tenure-of-title), Memorial Sloan-Kettering Cancer Center
New York, NY

2006-2007: Vice President, Laboratory Science
2007-2008: Vice President, Oncology and Research
2007-2008: Director, Curtis and Elizabeth Anderson Cancer Institute
2006-2008: Professor of Obstetrics and Gynecology, Surgery, Medicine, and Division of Basic Medical Sciences, Mercer University School of Medicine - Savannah
Assistant Dean for Research, Mercer University School of Medicine - Savannah
Distinguished Cancer Scholar, State of Georgia
Memorial University Medical Center, Savannah, GA

2008-2010: Senior Vice President and Chief Scientific Officer
Robert C. Young, MD, Chair in Cancer Research
Professor (with tenure), Women's Cancer Program
Fox Chase Cancer Center, Philadelphia, PA

2010-2014: Senior Vice President, Molecular Medicine
Robert C. Young, MD, Chair in Cancer Research
Executive Director, Cancer Genome Institute
Chief, Division of Molecular Pathology
Professor (with tenure), Cancer Biology Program
Fox Chase Cancer Center, Philadelphia, PA

2008-2015: Professor (with tenure), Cancer Biology Program
Robert C. Young, MD, Chair in Cancer Research
Fox Chase Cancer Center, Philadelphia, PA

2015-present: Professor (with tenure) and Chair, Department of Human and Molecular Genetics
Professor, Department of Obstetrics and Gynecology
Associate Dean for Basic Research and Graduate Programs
Herbert Wertheim College of Medicine
Florida International University
Miami, FL

2015-present: Associate Deputy Director, Translational Research and Genomic Medicine
Miami Cancer Institute
Baptist Health South Florida
Miami, FL

Scientific and Medical Societies:

American Association for the Advancement of Science (1982)
American Association for Cancer Research (1990)
American Society for Cell Biology (1992)
American Society of Clinical Oncology (2002)
American Society of Human Genetics (1997)
Association for Molecular Pathology (2014)
International Society of Gynecologic Oncology (2006)
Society of Gynecologic Oncology (1997)

Awards, Fellowships, and Grants:

Award for Special Achievement
Department of Health, Education, and Welfare, NIH, July, 1980.

Environmental Pathology Training Fellowship (Institutional NRSA)
NIH/NIEHS, T32-ES07017, March, 1986.

National Research Service Award (Individual)
NIH/NCI, F32-CA0524, February, 1988.

Co-Principal Investigator, Gynecologic Cancer Foundation/Karin Smith Award, "Gene Therapy of Ovarian Cancer" (Univ of Pennsylvania); 6/1/96-5/31/97; $50,000 total direct costs.

Principal Investigator, "Molecular Genetics of Gynecologic Cancers"
NIH/NCI, R01-CA67164; 10/1/96-9/30/00; $482,401 total direct costs.

Principal Investigator, "Genetic Mechanism of BRCA1-Linked Ovarian Tumorigenesis", NIH/NCI, R01-CA71840, 10/1/96-9/30/00; $465,563 total direct costs.

Principal Investigator, "Genetic Mechanism of BRCA-Linked Ovarian Tumorigenesis", NIH/NCI, R01-CA71840, 2/1/01-1/31/05; $676,000 total direct costs.

Principal Investigator, "Basic and Translational Research Program in the Molecular Genetics of Gynecologic and Breast Cancers: New Strategies for Prevention, Early Detection, and Treatment", Keck Foundation; 1/1/99-12/31/03; $2,500,000 direct costs.

Principal Investigator, "Molecular Classification of Ovarian Cancers", NIH/NCI, U01-CA88175; 10/1/00-9/30/05; $655,976 total direct costs.

Principal Investigator, "Preclinical Alterations in Breast Epithelium of BRCA Heterozygotes", Breast Cancer Research Foundation, 10/1/00; $170,000 total direct costs.

Principal Investigator, "Molecular Genetic Basis of Invasive Breast Cancer Risk Associated with Lobular Carcinoma in Situ", Breast Cancer Research Foundation, 10/1/01; $243,356 total direct costs.

Principal Investigator, "Prediction of Breast Cancer Risk by Gene Expression Profiling", Breast Cancer Alliance, 11/1/01; $130,000 total direct costs.

Principal Investigator, "Molecular Response to Selective Estrogen Receptor Modulators (SERMs) in Human Breast Cancer Cells", Breast Cancer Research Foundation, 10/1/02; $228,862 total direct costs.

Principal Investigator, "Genetic Polymorphisms and Risk of Breast Cancer", Breast Cancer Alliance, 11/1/02; $91,592 total direct costs.

Principal Investigator, "Somatic Genetic Alterations in *BRCA*-Linked Human Breast Cancer", Breast Cancer Research Foundation, 10/1/03; $230,000 total direct costs.

Principal Investigator, "Molecular Classification of Endometrial Cancers", NIH/NCI, R01-CA100272; 4/1/04-3/31/08; $1,350,000 total direct costs.

Principal Investigator, "Prediction of Breast Cancer Risk by Whole Genome Profiling", Department of Defense, CDMRP, BC033728; 8/1/04-7/31/05; $75,000 total direct costs.

Principal Investigator, "Prediction of Breast Cancer Risk by Whole Genome Profiling", Breast Cancer Research Foundation, 10/1/04; $250,000 total direct costs.

Project Director, "Project 1: Role of CA125/MUC16 in Ovarian Tumorigenesis", NIH/NCI, P01-CA52477-13, "Epithelial Ovarian Cancer Program Project"; 4/1/05-3/31/10; $7,374,628 total direct costs.

Co-Principal Investigator, "Polygenic Basis of Breast Cancer", Breast Cancer Research Foundation, 10/1/05; $250,000 total direct costs.

Georgia Distinguished Cancer Scholar, Georgia Cancer Coalition, 2006-2010; $750,000 total direct costs.

Principal Investigator, "Recruiting shRNA Functional Screening Expertise", Pennsylvania Department of Community and Economic Development Grant, C000043689, 1/1/09-6/30/10; $150,000 total costs.

Principal Investigator, American Cancer Society Institutional Research Grant, IRG-92-027-15, 1/1/08-12/31/10; $360,000 total costs.

Principal Investigator, "The Exomes of Ovarian Tumors of Low Malignant Potential and Low Grade Ovarian Cancers", Sandy Rollman Ovarian Cancer Foundation; 6/1/10-5/31/11; $60,000 direct costs.

Mentor, "Determine the Role of Canonical Wnt Signaling in Ovarian Tumorigenesis", CDMRP/DOD, Ovarian Academy Award W81XWH-10-1-0823 (PI: R Zhang), 9/15/10-3/29/13; $750,000 total direct costs.

Angela Carlino Excellence in Ovarian Cancer Research Award, Sandy Rollman Ovarian Cancer Foundation; October, 2010.

Principal Investigator, "The Transcriptome of Platinum Resistance in Ovarian Cancer", The Carpenter Foundation; 7/1/12-6/30/13; $50,000 total direct costs.

Principal Investigator, "FCCC-PENN SPORE in Ovarian Cancer", NIH/NCI, P50 CA083638; 8/21/09–5/31/15; $9,996,150 total direct costs.

Mentor, "Identifying Determinants of PARP Inhibitor Sensitivity in Ovarian Cancer", CDMRP/DOD, Ovarian Academy Award OC130212 (PI: N Johnson), 2/1/14-1/31/19; $750,000 total direct costs.

Rosalind Franklin Award for Excellence in Ovarian Cancer Research, Ovarian Cancer Research Fund Alliance; July, 2016.

Co-Investigator, "The Impact of Radiation Dose on Brain Morphology, Volumetric Changes, Endocrine Function, and Neurocognitive Function Following Cranial Radiation Therapy in Children with Brain and Skull Base Tumors", Florida Department of Health, Award 8LA04 (PI: M. Hall), 6/14/18-4/30/22; $700,000 total direct costs.

Editorial Positions:

1993-1997:	Associate Editor, *Molecular and Cellular Differentiation*
1994-2006:	Associate Editor, *Molecular Carcinogenesis*
1997-2003:	Editorial Board, *Gynecologic Oncology*
2003-2008:	Associate Editor, *Gynecologic Oncology*
2004-2008:	Editorial Board, *Journal of Clinical Oncology*
2004-2017:	Editorial Board, *American Journal of Pathology*
2017-present	Editorial Board, *Anticancer Research*

Committee Assignments (Previous):

Member, Task Force for Activities and Membership Development, American Association for Cancer Research, 1993.

Member, Epidemiology Committee, DOD Breast Cancer Program Review, 1994.

Member, Program Committee, Annual Meeting of the American Association for Cancer Research, 1995.

Member, Physiology Committee, DOD Gulf War Illness Program Review, 1995.

Member, Reproductive Biology Committee, DOD Women's Health Program Review, 1996.

Member, Special Review Group, "Endocrine Disrupting Chemicals and Women's Health Outcomes" (RFA 96-003), NIH/NIEHS, 1996.

Member, Epidemiology Committee, DOD Breast Cancer Program Review, 1996.

Invited Participant, American Cancer Society Workshop on Heritable Cancer Syndrome and Genetic Testing, 1996.

Member, Special Review Panel for Program Project Application P01-CA73992, "Molecular and Clinical Approaches to Colon Cancer Precursors", University of Utah, 1996.

Ad-Hoc Member, Program Committee, Society for Gynecologic Oncologists Annual Meeting, 1997.

Invited participant, "The Strategic Planning Conference on New Directions in Ovarian Cancer Research", The U.S. Public Health Service's Office on Women's Health, Washington, DC, 1997.

Member, Committee for DOD Ovarian Cancer Program Review, 1998.

Invited participant, "Implementation Meeting for New Directions in Ovarian Cancer Research", The National Cancer Institute and The Society of Gynecologic Oncologists, Bethesda, MD, 1998.

Member, Special Review Panel for National Cancer Institute Program Project Grant Application, "Epidemiologic and Genetic Studies of Breast Cancer", Mayo Foundation, Rochester, MN, February, 1999.

Ad Hoc Member, National Cancer Institute Scientific Review Group, Subcommittee E (Prevention and Control), Bethesda, MD, 1999.

Ad-Hoc Member, Initial Review Group, Small Grants Program for Cancer Epidemiology, National Cancer Institute, Bethesda, MD, 1999.

Ad-Hoc Member, Peer Review Committee on Molecular Genetics and Oncogenes, American Cancer Society, 1999.

Member, Specified Appropriations Program Peer Review Committee, United States Army Medical Research and Material Command, 1999.
Member, Committee for DOD Ovarian Cancer Program Review, 1999.

Member, Special Review Panel for National Cancer Institute Program Project Grant Application, "DNA Repair Genes and Cancer", University of Kentucky Medical Center, Lexington, KY, September, 1999.

Member, Program Committee, Society of Gynecologic Oncologists Annual Meeting, 2000.

Member, Special Review Panel for National Cancer Institute Program Project Grant Application, "Dietary and Hormonal Determinants of Cancer in Women" (Nurses' Health Study), Brigham and Women's Hospital, Boston, MA, February, 2000.

Invited Participant, Gynecologic Cancer Translational Research Retreat (GOG/NCI), Chantilly, VA; May, 2000.

Course Director, Second International Conference on Ovarian Cancer, Memorial Sloan-Kettering Cancer Center, New York, NY; June, 2000.

Invited Participant, Conference on Ovarian Cancer Screening, NCI, Bethesda, MD; September, 2000.
Member, Committee for DOD Ovarian Cancer Program Review, 2000.

Invited Participant, NCI Gynecologic Cancers Progress Review Group Roundtable Meeting, Herndon, VA; June, 2001.

Member, Committee for DOD Ovarian Cancer Program Review, 2001.

Ad-Hoc Member, PTHC/CAMP Scientific Review Group, National Institutes of Health, Washington, DC; June, 2002.

Member, Epidemiology Panel, DOD Breast Cancer Program Review, 2002.

Member, Special Review Panel for National Cancer Institute Program Project Grant Application, "Cervical Cancer: Biology of Initiation and Progression", Emory University, Atlanta, GA, September, 2002.

Member, Scientific Review Group for Ovarian SPORE Applications, National Cancer Institute, Bethesda, MD; June, 2003.

Invited participant, Borderline Ovarian Tumor Consensus Workshop, National Cancer Institute, Bethesda, MD; August, 2003.

Member, Special Emphasis Panel ZCA1 SRRB-4 J1 R, "Strategic Partnerships to Evaluate Cancer Signatures", National Institutes of Health, 2004.

Member, Program Committee, Society of Gynecologic Oncologists Annual Meeting, Miami Beach, FL; 2005.

Ad-Hoc Member, NCI Scientific Review Group, Subcommittee E – Cancer Epidemiology, Prevention, and Control, Bethesda, MD; April, 2005.

Chair, Special Emphasis Panel, ZRG1 ONC-U (03), Breast and Ovarian Cancer Genetics, Center for Scientific Review, National Institutes of Health; July, 2005.

Invited Participant, National Cancer Institute Ovarian Cancer State-of-the-Science Meeting, Bethesda, MD; September, 2005.

Member, Education Committee, Society of Gynecologic Oncologists, 2000-2004
Member, Institutional Review Board, Memorial Sloan-Kettering Cancer Center, 1999-2006.

Member, Human Tissue Utilization Committee, Memorial Sloan-Kettering Cancer Center, 2002-2006.

Member, Computational Biology Program Search Committee, Memorial Sloan-Kettering Cancer Center, 2002-2006.

Member, Database Working Group, Memorial Sloan-Kettering Cancer Center, 2002-2006.

Ad-Hoc Member, Committee on Appointments and Promotions, Memorial Sloan-Kettering Cancer Center; July 2002, October, 2003, April, 2004, March, 2005.\\

Member, Translational and Integrative Medicine Grant Review Committee, Memorial Sloan-Kettering Cancer Center; 2003-2006.

Member, Institutional Review Board Workflow Committee, Memorial Sloan-Kettering Cancer Center; 2004-2006.

Invited Participant, Joint NCI/British National Cancer Research Institute Gynecologic Cancer Intergroup Endometrial Cancer State-of-the-Science Meeting, Manchester, UK; November, 2006.

Member, Integration Panel, DOD Ovarian Cancer Research Program, 2001-2008.

Chair, Integration Panel, DOD Ovarian Cancer Research Program, 2005-2006.

Member, Peer Review Committee on Molecular Genetics and Oncogenes, American Cancer Society, 2002-2006.

Charter Member, Cancer Biomarkers Study Section, Center for Scientific Review, National Institutes of Health, 2003-2008.

Chair, Molecular and Cellular Biology and Genetics Peer Review Panel, Susan G. Komen for the Cure Grants Program; January, 2008.

Member, External Advisory Committee, SPORE in Ovarian Cancer, Fox Chase Cancer Center, Philadelphia, PA; 2003-2008.

Chair, Appointments and Promotions Committee, Anderson Cancer Institute, Memorial University Medical Center; 2006-2008.

Member, Board of Directors, Georgia Center for Oncology Research and Education; 2006-2008.

Member, Georgia Cancer Coalition Distinguished Cancer Scholar Review Committee; 2006-2008.

Chair, Medical Research Advisory Committee, Memorial University Medical Center; 2007-2008.

Member, Board of Advisors, College of Science and Technology, Georgia Southern University; 2006-2008.

Member, Special Emphasis Panel, NCI-ARRA P30 Biomedical Research Core Center Review, Rockville, MD; July, 2009.

Member, CDMRP Ovarian Cancer Grant Review Panel OC-4, Reston, VA; August, 2009.

Member, Scientific Advisory Committee, Ovarian Cancer Research Fund, 1999-2009.

Chair, DOD/CDMRP Breast Cancer Grant Review Panel MBG-B, Reston, VA; January, 2010.

Member, Scientific Review Group, NIH/NCI ZCA1 SRLB-R M1 R, Exceptional, Unconventional Research Enabling Knowledge Acceleration (EUREKA), Rockville, MD; March, 2010.

Member, Scientific Review Group, EDRN Biomarker Development Labs (U01), NIH/NCI ZCA1 SRLB-C M1 B, Bethesda, MD; May, 2010.

Chair, DOD/CDMRP Breast Cancer Research Program Grant Review Panel TRN-MBG, Reston, VA; May, 2010.

Chair, DOD/CDMRP Breast Cancer Research Program Grant Review Panel IDEA-MBG, Reston, VA; June, 2010.

Chairman, External Advisory Committee, SPORE in Ovarian Cancer, Dana-Farber/Harvard Cancer Center, Boston, MA; 2003-2010.

Member, Program Committee, 13th Biennial Meeting of the International Gynecologic Cancer Society, Prague, Czech Republic, 2010.

Member, Nominations Committee, Fox Chase Cancer Center, 2008-2010.

Member, Scientific Review Group, NIH/NCI ZCA1 SRLB-2 M1 R, Exceptional Unconventional Research Enabling Knowledge Acceleration (EUREKA), Bethesda, MD; March, 2011.

Member and Co-Chair, Subcommittee on Tissue Utilization, Gynecologic Oncology Group, 1997-2011.

Member, Scientific Review Group, NIH/NINR ZNR1 REV M 09, Personalized Genomics for Symptom Management: Bridging the Gaps from Genomic Discovery to Improved Health Outcomes, Bethesda, MD; June, 2011.

Member, Program Committee, Society for Gynecologic Oncology Annual Meeting, 2012.

Member, Board of Directors, Gynecologic Cancer Foundation (now Foundation for Women's Cancer); 2006-2013.

Member, Cancer Center Support Grant Executive Committee, Fox Chase Cancer Center, 2008-2013.

Member, President's Council, Fox Chase Cancer Center, 2008-2013.

Chair, DOD/CDMRP Breast Cancer Research Program Grant Review Panel BC12 TRN2, Reston, VA; February, 2013.

Member, Scientific Review Group, NCI ZCA1 RPRB-O (O1), NCI Small Grants Program for Cancer Research (NCI Omnibus R03), Reston, VA; June, 2013.

Member, Scientific Review Committee, DOD/CDMRP Ovarian Cancer Research Program Pilot Award Letter of Intent Review; July, 2013.

Chair, DOD/CDMRP Breast Cancer Research Program Grant Review Panel TRN2-CMB, Chantilly, VA; March, 2014.

Member, Executive Committee on Research, Fox Chase Cancer Center, 2008-2014.

Member, Scientific Review Committee, DOD/CDMRP Ovarian Cancer Research Program Pilot Award Letter of Intent Review; July, 2014.

Member, NCI Special Emphasis Panel for Review of Omnibus R21/R03 Applications in Response to PAR12-145/144; July, 2014.

Member, Scientific Review Committee, DOD/CDMRP Breast Cancer Research Program Grant Review Panel CBY-2, Reston, VA; July, 2014.

Member, Ovarian Cancer SPORE Executive Committee, Fox Chase Cancer Center, 2008-2015.

Founding Member, Genomic Advisory (Tumor) Board, Fox Chase Cancer Center, 2012-2015.

Member, Program Committee, Society of Gynecologic Oncology Annual Meeting, Chicago, IL; March, 2015.

Member, DOD/CDMRP Ovarian Cancer Research Program Pre-Application Review Panel, Pilot Award Mechanism; May-June, 2015.

Chair, DOD/CDMRP Breast Cancer Research Program Grant Review Panel, Molecular Biology and Genetics, Reston, VA; June, 2015.

Chair, Society of Gynecologic Oncology Genetics Delivery Care Summit, 2014-2015.

Invited Participant, Workshop on Ovarian Cancer, US Food and Drug Administration, White Oak, MD; July, 2015.

Member, Novartis Future of Diagnostic Laboratories Advisory Board, Austin, TX; November, 2015.

Invited Participant, Banbury Center Conference on, "Preventing BRCA-Related Cancer: a Think Tank for Innovative Strategies, Milestone Objectives, and Research Priorities", Cold Spring Harbor, NY; November, 2015.

Member, Committee on Experimental Medicine, Gynecologic Oncology Group (now NRG Oncology), 1997-2014.

Co-Chair, Banbury Center Conference on, "After UKCTOCS: Public Messaging on Screening and Early Detection of Ovarian Cancer", Cold Spring Harbor, NY; February, 2016.

Member, FORCE (Facing Our Risk of Cancer Empowered) Advisory Board; 2003-2013.

Member, Development Committee, Foundation for Women's Cancer, 2013-2015.

Member, National Cancer Institute Special Emphasis Panel/Scientific Review Group 2016/05 ZCA1 PCRB-C (C2) B - Cell and Animal Models for Researching Disparities; February, 2016.

Chair, DOD/CDMRP Ovarian Cancer Research Program Grant Review Panel, Pathobiology Pilot Award Program; September, 2016.

Member, Clinical Practice Committee, Society of Gynecologic Oncology, 2014-2017.

Member, AACR Clinical and Translational Cancer Research Grants Scientific Review Committee, 2015-2017.

Member, National Cancer Institute Clinical Translational R21 and Omnibus R03 Special Emphasis Panel ZCA1 SRB-P (O1); May, 2018.

Member, Medical Student Interview Panel, Herbert Wertheim College of Medicine, Florida International University; 2017-2018.

Member, National Cancer Institute Special Emphasis Panel, ZCA1 SRB-P (J1) – Clinical and Translational Exploratory/Developmental Studies; September, 2018.

Co-Chair, Banbury Center Conference on, "Towards a Cure for Advanced Ovarian Cancer", Cold Spring Harbor, NY; October, 2018.

Member, Scientific Advisory Committee, Ovarian Cancer Research Alliance, 2001-2018.

Chair, Scientific Advisory Committee, Ovarian Cancer Research Alliance, 2009-2018.

Member, Board of Directors, Ovarian Cancer Research Fund Alliance, 2012-2018.

Member, Scientific Review Committee, National Cancer Institute Specialized Programs of Research Excellence II (P50); 2019/05 ZCA1 RPRB-7 (M1) P; January, 2019.

Member, Special Emphasis Panel-5, National Cancer Institute Clinical and Translational R21 and Omnibus R03; 2019/05 ZCA1 SRB-P (M2) S; January, 2019.

Committee Assignments (Current):

Member, External Advisory Board, SPORE in Ovarian Cancer, MD Anderson Cancer Center, Houston, TX; 2009-present.

Vice-Chair, Joint Scientific Advisory Committee, Stand Up to Cancer (SU2C) Ovarian Cancer Dream Team Grant; 2014-present.

Member, Cancer Education Committee: Cancer Prevention, Hereditary Genetics, and Epidemiology Track, American Society of Clinical Oncology (ASCO); 2016-present.

Member, Clinical Scientific Review Committee, Miami Cancer Institute, 2016-present.

Member, Board of Directors, Society of Gynecologic Oncology, 2017-2020.

Member, Medical Student Interview Panel, Herbert Wertheim College of Medicine, Florida International University; 2018-2019.

Member, Board of Directors, Florida International University Research Foundation; 2017-present.

Invited Lectures (Since 1992):

"Cell structure and tumor suppression" and "Molecular genetic techniques in human cancer research." South American Course in Cancer Research; Caracas, Venezuela; February, 1992.

"Form and function in molecular carcinogenesis." Third Frontiers in Science Symposium; NIH/NIEHS, Research Triangle Park, NC; April, 1992.

"Expression and function of the DCC gene in neural differentiation." Gordon Research Conference on Cancer; Newport, RI; August, 1992.

"DCC gene expression and function." Fifth Conference on Differentiation Therapy; Sardinia, Italy; September, 1992.

"Tumor suppressor genes I" and "Tumor suppressor genes II." Department of Toxicology, North Carolina State University, Raleigh, NC; September, 1992.

"Molecular genetics of human endometrial carcinoma." Department of Pathology, University of North Carolina, Chapel Hill, NC; September, 1992.

"Methods for the study of molecular genetics in human cancer." Department of Pathology, Jikei University School of Medicine, Tokyo, Japan; October, 1992.

"The role of cell structure in tumor suppression." Fourth International Conference of Anticancer Research; Crete, Greece; October, 1992.

"Molecular markers and endometrial cancer." Department of Epidemiology, University of North Carolina School of Public Health, Chapel Hill, NC; November, 1992.

"Tumor suppressor genes." Department of Epidemiology, University of North Carolina, Chapel Hill, NC; November, 1992.

"Role of cell and tissue structure in tumor suppression." Department of Pathology, Johns Hopkins University School of Medicine, Baltimore, MD; November, 1992.

"Endometrial hyperplasia and adenocarcinoma: Molecular genetic characterization and determinants of risk." American Association of Pathologists Annual Meeting; New Orleans, LA; March, 1993.

"The environment and women's health." First Annual Environmental Careers Symposium; NIH/NIEHS, Research Triangle Park, NC; May, 1993.

"Cell structure and tumor suppression." Gordon Research Conference on Biological Structure and Gene Expression; Volterra, Italy; May, 1993.

"Molecular genetics of human endometrial carcinoma." Department of Molecular and Cell Biology, University of California at Berkeley, Berkeley, CA; May, 1993.

"Molecular genetics of human endometrial carcinoma." Gordon Research Conference on Hormonal Carcinogenesis; Newport, RI; August, 1993.

"Molecular genetics of endometrial hyperplasia." Workshop on Alternatives to Hysterectomy, National Institutes of Health, Bethesda, MD; May, 1994.

"Molecular genetics of ovarian carcinoma." Third International Symposium on Ovarian Function, Sapporo, Japan; September, 1994.

"Molecular genetics of estrogen-associated cancers." Conference on Molecular Mechanisms of Environmental Carcinogenesis, Research Triangle Park, NC; September, 1994.

“Molecular genetics of gynecologic cancers.” University of Pennsylvania Cancer Center Symposium on New Developments in Cancer Therapy: Focus on Gynecologic Cancers, Philadelphia, PA; December, 1994.

“Genetics and molecular medicine for the gynecologic oncologist”, “BRCA1 and other genes involved in hereditary predisposition to reproductive cancer”, Society of Gynecologic Oncologists Annual Meeting, San Francisco, CA; February, 1995.

“Hereditary Gynecologic Cancers.” Grand Rounds, Department of Obstetrics and Gynecology, University of Pennsylvania Medical Center, Philadelphia, PA; March, 1995.

“Endometriosis and the Environment: Biomarkers of Toxin Exposure.” Endometriosis 2000 Conference, National Institutes of Health, Bethesda, MD; May, 1995.

“Hereditary Gynecologic Cancers.” Grand Rounds, Department of Obstetrics and Gynecology, Medical College of Pennsylvania, Philadelphia, PA; May 1995.

“E-Cadherin as a Tumor Suppressor.” Gordon Research Conference on Cell Contact and Adhesion, Andover, NH; June, 1995.

“Mismatch Repair.” American Urologic Association Summer Research Conference, Houston, TX; August, 1995.

"Genetic Characterization of Human Endometrial Carcinoma." Ninth International Conference on Carcinogenesis and Risk Assessment, Austin, TX; November, 1995.

"Molecular Genetics of Ovarian Carcinoma." The Finnish Medical Society Duodecim Annual Meeting, Turku, Finland; November, 1995.

"Hereditary Gynecologic Cancers." Department of Pathology Grand Rounds, University of Pennsylvania Medical Center, Philadelphia, PA; November, 1995.

"Genetics of Hereditary Breast and Gynecologic Cancers." Postgraduate Course on Molecular Biology of Gynecologic Cancers: Clinical Implications for the 1990s. Society of Gynecologic Oncologists Annual Meeting, New Orleans, LA; February, 1996.

"Molecular Genetics of Hereditary Gynecologic Cancers." Department of Obstetrics and Gynecology Grand Rounds, Thomas Jefferson University, Philadelphia, PA; February, 1996.

"Hereditary Nonpolyposis Colorectal Cancer: Ethical, Legal, and Social Implications of Genetic Testing and Counseling for High Risk Individuals." American Radium Society Annual Meeting, San Francisco, CA; March, 1996.

"Molecular Genetics of Hereditary Gynecologic Cancers." Department of Genetics, University of Pennsylvania, Philadelphia, PA; May, 1996.

"Molecular Genetics of Hereditary Endometrial and Ovarian Carcinomas." President's Symposium of the New York Pathological Society, New York, NY; June, 1996.

"Familial Ovarian Cancer: Laboratory Diagnosis." Current Concepts in Women's Health Care: Seventeenth Annual Postgraduate Course, University of Pennsylvania Medical Center, Philadelphia, PA; June 1996.

"Molecular Genetics of Hereditary Gynecologic Cancers." Barbara Moore Jordan Visiting Professorship, Memorial Sloan-Kettering Cancer Center, New York, NY; July 1996.

"Breast Cancer Genetics." Keynote Lecture at the First Annual New Jersey Breast Cancer Research Symposium, Princeton, NJ; October, 1996.

"Estrogen as a Human Carcinogen: Molecular Genetics of Gynecologic Cancers." US-Japan Cooperative Medical Science Program, Environmental Mutagenesis and Carcinogenesis Panel, Tokyo, Japan; November, 1996.

"Hereditary Breast and Ovarian Cancer: Molecular Genetics and Clinical Implications." Grand Rounds, Department of Surgery, Memorial Sloan-Kettering Cancer Center, New York, NY; December, 1996.

"Molecular Genetics of Hereditary Gynecologic Cancers." Solid Tumor Oncology Conference, Department of Medicine, Memorial Sloan-Kettering Cancer Center, New York, NY; February, 1997.

"Molecular Genetics of Hereditary Ovarian Cancer." Basic Science Postgraduate Course; "BRCA1/2 and Other Genes Involved in Hereditary Predisposition to Ovarian Cancer." Breakfast Session, Society of Gynecologic Oncologists Annual Meeting, Phoenix, AZ; March, 1997.

"Genetics of Ovarian Cancer." Helene Harris Memorial Trust 6th International Forum on Ovarian Cancer, Los Angeles, CA; May, 1997.

"Genotype-Phenotype Correlations in Hereditary Ovarian Cancer." Symposium on Ovarian Cancer: Prevention, Genetics and Treatment Challenges, Toronto, Ontario; May, 1997.

"Molecular Genetics of Hereditary Gynecologic Cancers." Department of Pathology Grand Rounds, Memorial Sloan-Kettering Cancer Center, New York, NY; July, 1997. "Quantitative Methods in Cancer Genetics."

Cancer Genetic Counseling and Testing: A Multidisciplinary Course, The Sarah Lawrence College Human Genetics Program, New York, NY; July, 1997.

"Hereditary Gynecologic Cancers: Molecular Genetics and Clinical Implications." 26th Congress of Gynecologic Pathology and Colposcopy, Tokyo, Japan; July, 1997.

"Molecular Genetics of Estrogen-Associated Human Cancers." Gordon Research Conference on Hormonal Carcinogenesis, Tilton, NH; July, 1997.

"Basic Principles of Genetics for Practicing Clinicians", Genetic Techniques - Relevance for Practicing Clinicians", and "Genetics of Gynecologic Sarcomas and Clinical Implications". European School of Oncology Conference on Molecular Genetics in Gynecologic and Breast Cancer and Its Clinical Implications: Bridging the Gap, Budapest, Hungary; November, 1997.

"Studies on the Molecular Mechanism of Estrogen-Associated Human Cancers." Department of Biochemistry, Mount Sinai University School of Medicine, New York, NY: November, 1997.

"Molecular Genetics of Hereditary Gynecologic and Breast Cancers." Distinguished Lecturer in Oncology, University of Texas M.D. Anderson Cancer Center, Houston, TX; January, 1998.

"Genetics of Hereditary Gynecologic Cancers: What patients are asking their gynecologists." Obstetrical Society of Philadelphia, Philadelphia, PA; February, 1998.

"Molecular Genetics of Hereditary Gynecologic Cancers." Grand Rounds, Department of Obstetrics and Gynecology, Allegheny University of the Health Sciences, Philadelphia, PA; February, 1998.

"Endometrial Cancer." Course on Human Genetics and Human Cancer, Memorial Sloan-Kettering Cancer Center, New York, NY; May, 1998.

"Molecular Genetics of Hereditary Gynecologic Cancers: Clinical Implications." New York Gynecology Society, New York, NY; May, 1998.

"Hereditary Ovarian Cancer: Molecular Genetics and Clinical Implications." IVth Sapporo International Symposium on Ovarian Function, Sapporo, Japan; August, 1998.

"Molecular Pathogenesis of Endometrial Neoplasia." Grand Rounds, Department of Pathology, Brigham and Women's Hospital, Boston, MA; October, 1998.

"Hereditary Gynecologic Cancers: Molecular Genetics and Clinical Implications." Visiting Professor Program, Department of Pathology, Montefiore Medical Center, Bronx, NY; October, 1998.

"Molecular Genetics of Hereditary Gynecologic Cancers." Memorial Hospital Annual Alumni Meeting, Memorial Sloan-Kettering Cancer Center, New York, NY; November, 1998.

"Clinical and Pathologic Features of BRCA-Associated Hereditary Ovarian Cancers." Grand Rounds, Department of Surgery, Memorial Sloan-Kettering Cancer Center, New York, NY; November, 1998.

"Genetic Epidemiology of Ovarian Cancer." International Conference on Ovarian Cancer, The University of Texas M.D. Anderson Cancer Center, Houston, TX; February, 1999.

"Ovarian Cancer." Course on Human Genetics and Human Cancer, Memorial Sloan-Kettering Cancer Center, New York, NY; April, 1999.

"Genetics of Ovarian Cancer." Annual Conference of the National Corporate Medical Associates, Memorial Sloan-Kettering Cancer Center, New York, NY; June, 1999.

"Molecular Genetics of Hereditary Gynecologic Cancers." Scientific Symposium for Semi-Annual Business meeting of the Gynecologic Oncology Group, Scottsdale, AZ; July, 1999.

"Genetics." Breast Cancer Core Course, Memorial Sloan-Kettering Cancer Center, New York, NY; July, 1999.

"Genetic Susceptibility to Gynecologic Cancers." Cancer Smart Lecture Series, Memorial Sloan-Kettering Cancer Center, New York, NY; October, 1999.

"Molecular Genetics of Hereditary Breast Cancer: Clinical Implications." New York Pathological Society, New York, NY; February, 2000.

"Genetics of Hereditary Gynecologic Cancers." Postgraduate Course at the Society of Gynecologic Oncologists Annual Meeting, San Diego, CA; February, 2000.

"Molecular Genetics of Breast and Gynecologic Cancers." Course on Molecular Oncology, New York University School of Medicine, New York, NY; March, 2000.

Session Chair, Conference on Gynecologic Care of the Cancer Patient, Memorial Sloan-Kettering Cancer Center, New York, NY; March, 2000.

"Molecular Genetic Mechanism of Estrogen-Associated Human Tumorigenesis." Memorial Sloan-Kettering Cancer Center Scientific Retreat, March, 2000.

"Biology of Ovarian Cancer." Disease Management Team Conference Series (Gynecology), Memorial Sloan-Kettering Cancer Center, New York, NY; March, 2000.

"Preclinical Molecular Genetic Alterations in Breast and Ovarian Epithelium of BRCA Heterozygotes." American College of Surgeons Oncology Group Planning Conference. Memorial Sloan-Kettering Cancer Center; April, 2000.

Session Chair, Molecular Biology of Gynecologic Cancers, American Association for Cancer Research Annual Meeting, San Francisco, CA; April, 2000.

"Genetics of Hereditary Ovarian Cancer." Education Session on Ovarian Cancer, American Society of Clinical Oncology Annual Meeting, New Orleans, LA; May, 2000.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Ovarian Cancer National Alliance Third Annual Advocacy Conference, Washington, DC; September, 2000.

"Genetics of Cancer." Grand Rounds, Department of Medicine, Mercy Medical Center, Rockville Centre, NY; October, 2000.

"Can Molecular Markers Improve Risk Factor Determinations and Thereby Dictate Treatment and Improve Survival?", Plenary Session on Endometrial Cancer, VIII Meeting of the International Gynecologic Cancer Society, Buenos Aires, Argentina; October, 2000.

"Hereditary Ovarian Cancer: What We Know." Helene Harris Memorial Trust 8th International Forum on Ovarian Cancer, Houston, TX; March, 2001.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Gusberg Distinguished Lectureship in Gynecologic Oncology, Mt. Sinai Medical Center, New York, NY; April, 2001.

"Breast and Ovarian Cancers: Basic Science." A Comprehensive Review of Clinical Cancer Genetics, American Society of Clinical Oncology Annual Meeting, San Francisco, CA; May, 2001.

"Molecular Genetics of Hereditary Gynecologic and Breast Cancers: Clinical Implications." Grand Rounds, Department of Medicine, St. Clare's Medical Center, NJ; May, 2001.

"Molecular Biology of Gynecologic Cancers: Clinical Applications." Speaker of the Royal College of Physicians and Surgeons of Canada, Society of Gynecologic Oncologists of Canada Annual Meeting, St. John's, Newfoundland, Canada; June, 2001.

"Molecular Genetics of Hereditary Ovarian Cancer: Clinical Applications." Canadian Federation of Biological Sciences Annual Meeting, Ottawa, Canada; June, 2001.

"Molecular Genetics of Hereditary Ovarian Cancer: Translational Applications." NCI/Center for Cancer Research Grand Rounds, Bethesda, MD; July, 2001.

"Molecular Genetics of Hereditary Gynecologic Cancers: Clinical Implications. Grand Rounds, Department of Obstetrics and Gynecology, Long Island Hospital, Brooklyn, NY; October, 2001.

"Can Clinical Problems in Ovarian Cancer be Solved in the Laboratory?" Visiting Professorship, Department of Obstetrics and Gynaecology, University of Toronto, Toronto, Canada; October, 2001.

"Molecular Genetics of Hereditary Gynecologic and Breast Cancers: Clinical Implications." Grand Rounds, Department of Obstetrics and Gynecology, Columbia University, New York; March, 2002.

"Molecular Genetics of Hereditary Gynecologic Cancers." Postgraduate Course of Clinical Usefulness of Genetic Testing in Gynecologic Oncology. Society of Gynecologic Oncologists Annual Meeting, Miami Beach, FL; March, 2002.

"Cancer Genetics." Course on Molecular Oncology, New York University, New York; March, 2002.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Conference on Ovarian Cancer and High-Risk Women: Implications of Screening, Prevention, and Early Detection, University of Pittsburgh, Magee-Women's Hospital, Pittsburgh, PA; May, 2002.

"Basic Science of Breast and Ovarian Cancer." Comprehensive Course on Clinical Cancer Genetics, American Society of Clinical Oncology Annual Meeting, Orlando, FL, May, 2002.

"Toward a Molecular Classification of Endometrial Carcinoma." Education Session on Endometrial Carcinoma, American Society of Clinical Oncology Annual Meeting, Orlando, FL; May, 2002.

"Hereditary Gynecologic Cancers: Clinical Implications." National Corporate Medical Associates Annual Meeting, Memorial Sloan-Kettering Cancer Center, New York, NY; June, 2002.

"Molecular Genetics of Hereditary Ovarian Cancer." Third Annual International Conference on Ovarian Cancer, MD Anderson Cancer Center, Houston, TX; September, 2002.

"Molecular Biology of Ovarian Cancer: From Pathogenesis to Treatment." Symposium on Ovarian Cancer, International Gynecologic Cancer Society Biennial Meeting, Seoul, Korea; October, 2002.

"Histogenesis of Ovarian Cancer." The Ethel N. Ruvelson Lecture in Ovarian Cancer, 33rd Annual Autumn Seminar in Obstetrics and Gynecology, University of Minnesota, Minneapolis, MN; October, 2002.

"Hereditary Gynecologic Cancers: What We Know." Society of Gynecologic Oncologists Winter Meeting, Breckenridge, CO; March, 2003.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Helene Harris Memorial Trust 9th Biennial Forum on Ovarian Cancer, Stratford-upon-Avon, United Kingdom; March, 2003.

"Cáncer de Ovario: Historia Natural y Biología Molecular." Cánceres de Próstata, Mama y Ovario: Tumores Hormono-Dependientes, Universidad Internacional Menéndez Pelayo, Santander, Spain; July, 2003.

"Gynecologic Tumors." Session on New Directions in Cancer, AACR Annual Meeting, Washington, DC; July, 2003.

"Molecular Genetics of Hereditary Gynecologic and Breast Cancers: Clinical Implications." Hoag Cancer Center Grand Rounds, Newport Beach, CA; July, 2003.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Grand Rounds, Department of Pathology, Yale-New Haven Hospital, New Haven, CT; September, 2003.

"Gene Silencing by Estrogen Receptor-Dependent Promoter Methylation." e.hormone 2003, 5th Annual Conference on Environmental Estrogens. Tulane University, New Orleans, LA; October, 2003.

"Genetics of Hereditary Breast and Gynecologic Cancers: Clinical Implications." 5th Annual Kimmel Cancer Center Hereditary Cancer Conference. Thomas Jefferson University, Philadelphia, PA; November, 2003.

Distinguished Visiting Professorship. "Genetic Analysis of Ovarian Carcinoma Histogenesis. Department of Pathology, Johns Hopkins University, Baltimore, MD; November, 2003.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." 19th Annual Ella T. Grasso Memorial Conference. University of Connecticut Health Center, Hartford, CT; November, 2003.

The 13th Annual Per Kolstad Memorial Lecture. "Genetics of Hereditary Ovarian Cancer: Clinical Implications." The Norwegian Radium Hospital, Oslo, Norway; December, 2003.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Medical Oncology and Ovarian Cancer Research Program Seminar Series, Fox Chase Cancer Center, Philadelphia, PA; January, 2004.

"BRCA - A Paradigm for Hereditary Cancer Predisposition." Postgraduate Course on "Genetics for Gynecologic Oncologists", Society for Gynecologic Oncologists Annual Meeting, San Diego, CA; February, 2004.

"Role of Gene Expression Profiling in Distinguishing Biologically and Clinically Distinct Subclasses of Endometrial Carcinoma." Gynecologic Cancer Models, Mouse Models of Human Cancers Consortium (NCI) Meeting, San Juan, Puerto Rico; February, 2004.

"Human Cancer Genetics." Course on Molecular Oncology, New York University, New York, NY; February, 2004.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Mayo Oncology Society, Rochester, MN; March, 2004.

"Ovarian Cancer - New Concepts in Organ-Site Research." American Association for Cancer Research Annual Meeting, Orlando, FL; March, 2004.

"Insights into Biology and Clinical Behavior of Endometrial Carcinoma through Comprehensive Gene Expression Profiling." Symposium on Ovarian Cancer and Other Gynecologic Malignancies, New York, NY; April, 2004.

"Genetics of Hereditary Gynecologic Cancers." American Society of Clinical Oncology Annual Meeting, ASCO/SGO Special Session on Clinical Management of Patients with Hereditary Predisposition to Gynecologic Cancers, New Orleans, LA; June, 2004.

"Gene Silencing through Estrogen Receptor Mediated Promoter Methylation." Gordon Research Conference on Reproductive Tract Biology, New London, CT; June, 2004.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Third Early Detection Research Network Scientific Workshop, Bethesda, MD; June, 2004.

"Stratification of Intermediate Risk Disease by Gene Expression Profiling." 2nd Annual Uterine Cancer Biology Symposium, MD Anderson Cancer Center, Houston, TX; September, 2004.

"Is There a Molecular Basis for the Developmental Estrogenization Syndrome?" e.hormone 2004 Conference, New Orleans, LA; October, 2004.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Grand Rounds, Dana-Farber/Massachusetts General Hospital, Boston, MA; November, 2004.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Grand Rounds, Curtis and Elizabeth Anderson Cancer Institute at Memorial Health University Medical Center, Savannah, GA; December, 2004.

Chair, "Postgraduate Course on Molecular Biology for Gynecologic Oncologists." Society for Gynecologic Oncologists Annual Meeting, Miami Beach, FL; March, 2005.

"Genetics of the Early Natural History of Ovarian Cancer." Helene Harris Memorial Trust 10th Annual Biennial International Forum on Ovarian Cancer, Washington, DC; April, 2005.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Elkin Cancer Biology Seminar Series, Winship Cancer Institute, Emory University School of Medicine, Atlanta, GA; March, 2005.

"Microarray Technology in Gynecologic Cancer Research." 2nd International Symposium on Ovarian Cancer and Other Gynecologic Malignancies, New York, NY; April, 2005.

"Hereditary Ovarian Cancer." Postgraduate Course on Gynecologic Cancer 2005, Medical College of Georgia/Curtis and Elizabeth Anderson Cancer Institute, Savannah, GA; April, 2005.

"Role of Defective DNA Repair in Gynecologic Tumorigenesis." Lynne Cohen Symposium on the Emerging Role of Screening and Prevention in Women's Cancers, NYU University School of Medicine, New York, NY; April, 2005.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Multidisciplinary International Conference on Gynecologic Cancer, Bologna, Italy; June, 2005.

"Gene Silencing through Estrogen Receptor-Mediated Promoter Hypermethylation." Biomedical Research Seminar Program, Mercer University School of Medicine, Macon GA; September, 2005.

"Treatment of Hereditary Ovarian Cancer: Clinical and Experimental Approaches." And "Haploinsufficiency: Is it Important?" International Symposium on *BRCA*: Today and Tomorrow, Montréal, Canada; October, 2005.

"Opening Key Note Address: Genetic Analyis of Ovarian Carcinoma Histogenesis." Symposium on Ovarian Cancer: Prevention and Detection of the Disease and its Recurrence. University of Pittsburgh Cancer Institute, Pittsburgh, PA; October, 2005.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Grand Rounds, Department of Pathology and Laboratory Medicine, MD Anderson Cancer Center, Houston, TX; January, 2006.

"Cancer Genetics." Course on Molecular Oncology, New York University School of Medicine, New York, NY; March, 2006.

"Genome-Based Laboratory Approaches to Advancing the Practice of Gynecologic Oncology." Postgraduate Course on Translational Research, Society for Gynecologic Oncologists Annual Meeting, Palm Springs, CA; March, 2006.

"Translational Research." Memorial Health University Medical Center First Resident Alumni CME Program, Savannah, GA; June, 2006.

"Molecular Medicine." Department of Internal Medicine, Memorial Health University Medical Center, Savannah, GA; August, 2006.

"Molecular Basis of Improved Survival in *BRCA*-Linked Ovarian Cancers." 11th Bienniel Meeting of the International Gynecologic Cancer Society, Santa Monica, CA; October, 2006.

"Genetic Analysis of Ovarian Carcinoma Histogenesis." Winter Symposium, Department of Obstetrics and Gynecology, Rambam Health Care Campus, Haifa, Israel; January, 2007.

"Functional Analysis of the CA125 (MUC16) Gene Product in Ovarian Tumorigenesis." Helene Harris Memorial Trust 11th Bienniel International Forum on Ovarian Cancer, Lake Como, Italy; March, 2007.

Discussant, Focused Plenary Session on Translational Research in Ovarian Cancer. Society of Gynecologic Oncologists Annual Meeting, San Diego, CA; March, 2007.

"Innovative Cancer Research Activities in Georgia." Georgia Cancer Summit, Atlanta, GA; January, 2008.

"Applications of Genomics/Proteomics Technologies to Gynecologic Cancers?" Gynecologic Oncology Group Scientific Session on "Genomics and Proteomics: The Future is Now". GOG Semi-Annual Meeting, San Diego, CA; January, 2008.

"Molecular Evolution of Ovarian Cancer." 1st Ovarian Cancer Action International Conference, London, United Kingdom; March, 2008.

"Genetics 101." Sunrise Postgraduate Session, Society of Gynecologic Oncologists Annual Meeting, Tampa, FL; March, 2008.

Discussant, Focused Plenary Session on Translational Research, Society of Gynecologic Oncologists Annual Meeting, Tampa, FL; March, 2008.

"Genetic Profiling of Endometrial Cancers." Fifth International Symposium on Ovarian Cancer and Gynecologic Malignancies, New York, NY; March, 2008.

"Cancer Genetics." Grand Rounds, Department of Internal Medicine, Memorial University Medical Center, Savannah, GA; April, 2008.

"The Future of Healthcare: Genetic Medicine." Annual Meeting of the Coastal Empire Health Underwriters Association, Savannah, GA; May, 2008.

"Relevance of Tumor Biology to Prevention and Diagnosis." International Symposium on Hereditary Breast and Ovarian Cancer: Risks and Challenges, Bari, Italy; September, 2009.

"Whence Epithelial Ovarian Carcinoma?" Robert F. Ozols Symposium on Gyn Cancer: Gyn Cancers – the Next 25 Years, Philadelphia, PA; September, 2009.

Session Chair. Opening Plenary Session I; Interactive Session: "Hereditary Gynecologic Cancers." 13th Bienniel Meeting of the International Gynecologic Cancer Society, Prague, Czech Republic; October, 2010.

"Whence Epithelial Ovarian Carcinoma?" Ovarian Cancer National Alliance Regional Symposium; Radnor, PA; November, 2010.

"The Origin of Epithelial Ovarian Carcinoma: New Insights." Omniprex 2011 Ovarian Cancer Course; Philadelphia, PA; April, 2011.

"Whence Epithelial Ovarian Carcinoma?" Grand Rounds, Department of Obstetrics and Gynecology, Michigan State University School of Medicine; Grand Rapids, MI; May, 2011.

"Low Grade Serous Carcinomas." From Molecular Information to Cancer Medicine - NCI Translational Science Meeting 2011, Washington, DC; July, 2011.

"The Vision and the Reality: One Cancer Center's Journey toward Genomic Medicine." Keynote Session, The Clinical Genome Conference, San Francisco, CA; June, 2012.

"The Vision and the Reality: One Cancer Center's Journey toward Genomic Medicine." Keynote Session, Ion Torrent User's Group Meeting, Baltimore, MD; March, 2013.

"Cancer Genetics and the Evolution of Precision Medicine." Memorial Sloan-Kettering Cancer Center, New York, NY; May, 2013.

Co-Organizer, "Ovarian Cancer: Developing Research-Based Public Messaging on Early Detection and Screening." The Banbury Center, Cold Spring Harbor, NY; October, 2013.

"Cancer Genetics and the Evolution of Precision Medicine." Grand Rounds, Department of Obstetrics and Gynecology, New York University School of Medicine, New York, NY; February, 2014.

"Defective Homologous Recombination and Therapeutic Opportunities in Ovarian Cancer." First Annual Meeting of International Ovarian Cancer Consortium: Tumor Microenvironment and Drug Discovery, University of Oklahoma Health Sciences Center, Oklahoma City, OK; February, 2014.

"Ethical, Legal, and Social Implications of Clinical Next-Generation Sequencing." Cancer Prevention and Control Program, Fox Chase Cancer Center, Philadelphia, PA; March, 2014.

"Lecturette: The Use of "omics"-Based Predictors in Clinical and Translational Research." Society of Gynecologic Oncology Annual Meeting, Tampa, FL; March, 2014.

"Genetic Solutions to the Cancer Problem: A Personal Perspective." The Jackson Laboratory for Genomic Medicine, Farmington, CT; August, 2014.

Keynote Presentation: "The Vision and the Reality: One Cancer Center's Journey toward Genomic Medicine." Seventh Annual Predictive Cancer Biomarkers Conference, Washington, DC; August, 2014.

"The Vision and the Reality: One Cancer Center's Journey toward Genomic Medicine." Third Annual Genomics in Medicine Symposium – Molecular Medicine Tri-Conference 2015, San Francisco, CA; February, 2015.

Panel Member, "Targeted Oncology". BIO 2015 International Conference, Philadelphia, PA; June, 2015.

"The Vision and the Reality: One Cancer Center's Journey toward Genomic Medicine." 8th Annual Predictive Cancer Biomarkers Conference, Washington, DC; August, 2015.

"Cancer Genetics and the Evolution of Precision Medicine." Grand Rounds, Broward Health Medical Center, Ft. Lauderdale, FL; March, 2016.

"Genetics of Women's Cancers: Advances through Genomic Medicine." Fifth Annual Omar Pasalodos, MD, Memorial Lecture, Miami, FL; April, 2016.

"Advances in Genomic Medicine: Focus on Head and Neck Cancers." Fifth Annual Head and Neck Cancer Symposium, Miami, FL; April, 2016.

"Cancer Genetics in the Primary Care Setting." The International Symposium on Primary Care, Miami Beach, FL; July, 2016.

"Genomic Predisposition to Breast Cancer." Fourth Annual John M. Cassel, MD, Memorial Breast Cancer Symposium, Miami, FL; September, 2016.

"Updates on the UKCTOCS Trial." Ovarian Cancer State-of-the-Art Conference, Memorial Sloan-Kettering Cancer Center, New York, NY; October, 2016.

"Genetics of Cancer: New Opportunities through Genomic Medicine." Miami Medical Forum, Miami, FL; October, 2016.

"Cancer Genetics and the Evolution of Precision Medicine." Presidential Plenary Session, International Gynecologic Cancer Society Biennial Meeting, Lisbon, Portugal; October, 2016.

"Genetic Predisposition to Cancer." Baptist Health South Florida Research Summit: Bringing Cancer Research to the Community, Miami, FL; November, 2016.

"Germline Testing Meets Genomic Testing: How to Sort It Out." Second Annual West Cancer Center Oncology Conference: Collaboration for the Future Cure: Precision Medicine and Immuno-Oncology, Memphis, TN; November, 2016.

"Genetics of Women's Cancers: Advances through Genomic Medicine." Second Annual MSK Cancer Alliance Scientific Symposium, Miami, FL; January, 2017.

"Precision Medicine in Cancer Care: Global Challenges and Opportunities." Enmore Bio Conference, Nanjing, China; February, 2017.

"Genetics of Women's Cancers: Advances through Genomic Medicine." Grand Rounds, Department of Obstetrics and Gynecology, Lehigh Valley Health Network, Allentown, PA; May, 2017.

"How to Interpret Tumor Genomics for the Oncologist." Education Session on Cascade Testing: What to Do When Ascertaining Germline Mutations from Tumor and Other Genomic Testing. American Society of Clinical Oncology Annual Meeting, Chicago, IL; June, 2017.

"Genetics of Women's Cancers: Advances through Genomic Medicine." President's Guest Speaker, Miami Obstetrical and Gynecological Society, Miami, FL; September, 2017.

"Genetics of Women's Cancers: Advances through Genomic Medicine." Grand Rounds, Simon Cancer Center, Indiana University, Indianapolis, IN; October, 2017.

"Genomics and Pediatric Malignancies." Kids with Cancer Symposium, Miami, FL; December, 2017.

"The Challenges and Rewards for Bringing AI into the Clinic for Health and Disease Management." Panel Discussion, Precision Medicine World Conference, Mountain View, CA; January, 2018.

"Genomics Revolution in Cancer Care." Al and Janie Nahmad Speaker Series: Thought Leaders in Medicine, Miami, FL; April, 2018.

"Cancer Genomics." Baptist Health International Videoconference, Miami, FL; September, 2018.

"Estrogen and Cancer." Visiting Professorship, Department of Obstetrics and Gynecology, University of Chicago, Chicago, IL; September, 2018.

"BRCA, Genetics, and Genomics: Role in Ovarian Cancer." Fight N Heal Teal Symposium, Miami, FL; October, 2018.

Ad Hoc Reviewer:

American Journal of Human Genetics
American Journal of Obstet and Gynecol
American Journal of Pathology
Annals of Surgical Oncology
BBA Reviews on Cancer
BMC Cancer
Breast Cancer Research and Treatment
British Journal of Cancer
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Journal of the American Medical Association
Journal of Clinical Investigation
Journal of Experimental Medicine
Journal of Medical Genetics
Journal of Molecular Diagnostics
Journal of Molecular Endocrinology
Journal of the National Cancer Inst
Lancet
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Nature
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EXHIBIT B

Testifying History for Jeff Boyd, Ph.D.

University of Miami v. Agency for Health Care Administration and Baptist Hospital of Miami, Inc.
State of Florida Division of Administrative Hearings
Case No. 16-001698CON

University of Miami v. Baptist Hospital of Miami, Inc., and Agency for Health Care Administration
State of Florida Division of Administrative Hearings
Case No. 17-005301CON